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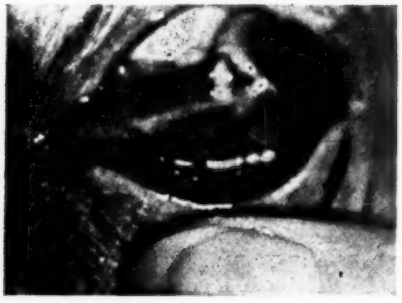

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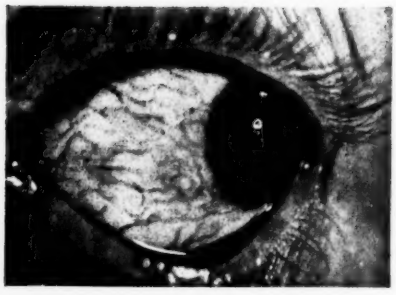

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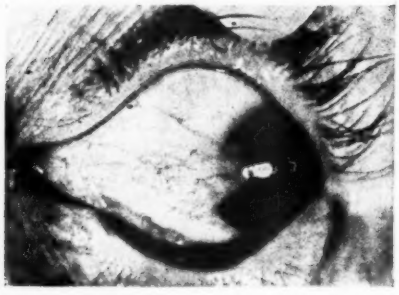

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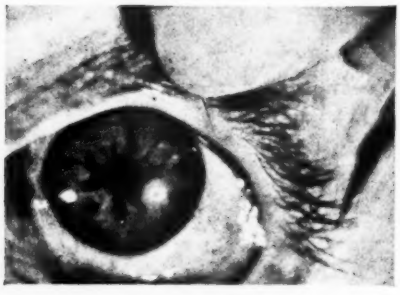

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FIG. 1 (HILGARTNER, WILSON, AND WILSON). (A) LARGE, EXTREMELY VASCULAR TYPE OF PTERYGIUM. (B) THIS SHOWS THE HEAD OF THE PTERYGIUM TRANSPLANTED BENEATH THE BULBAR CONJUNCTIVA BELOW. (C) THIN, RELATIVELY AVASCULAR TYPE OF PTERYGIUM. (D) THIS EYE HAD BEEN OPERATED ON SOME MONTHS BEFORE THIS PICTURE WAS TAKEN. IT SHOWS VASCULARITY AND THICKENING OF THE BULBAR CONJUNCTIVA. NO X-RAY THERAPY WAS GIVEN. (E) THIS EYE SHOWS A SLIGHT AMOUNT OF INJECTION FOLLOWING SURGERY AND X-RAY THERAPY.

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SURGICAL TREATMENT OF PTERYGIA FOLLOWED BY X-RAY THERAPY TO PREVENT RECURRENCE*

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One of the most common conditions seen in the southwest section of this country is that of a pterygium encroaching on the cornea of one or both eyes. As everyone knows, this is usually a painless, slow-growing, disfiguring growth composed of fibrillar connective tissue and epithelium of the conjunctiva. It may or may not be vascular. It is most commonly seen growing from the nasal side, but it may also grow from the temporal side at the same time, and, in some rare cases, the pterygium will descend from above, or ascend from below. This latter condition is, however, infrequent.

STRUCTURE OF PTERYGIUM

Doherty has divided the pterygium into four parts: (1) the cap, which is the flat grayish section which precedes the head as it encroaches on the cornea, (2) the head, (3) the neck, and (4) the body. The head consists of loose connective tissue which advances between Bowman's membrane and the epithelium, the latter becoming detached. Bowman's membrane is partly or completely destroyed. The neck and body consist of blood vessels, connective tissues, and conjunctival epithelium.

It is not our purpose to give a long dissertation on the pathology, incidence, and surgical technique for the removal of pterygia, but it is necessary to review briefly the facts. In conclusion we wish to present our technique for the prevention of the recurrence of the growth.

TYPES OF PTERYGIA

We feel that there are two distinct types of pterygia: (1) The small, slow growing, rather avascular pterygium with a flat, more or less whitish-gray head, and practically no cap. (2) The highly vascular, thick, somewhat inflamed growth, with elevated head and a very definite cap which extends some distance beyond the head onto the cornea. The second type is fast growing and invades the deeper structures of the cornea. If allowed to stay on the cornea for any length of time, a definite grayish scar may form beneath it, which is disfiguring after removal of the pterygium from the cornea.

It is this second type of pterygia with which we are really concerned and which usually recurs following surgery. The usual story is that the growth recurred soon after surgery was performed, sometimes within a few days after the operation, and that the growth developed more rapidly than it formerly had.

A true pterygium develops and grows without previous corneal ulceration or injury. A false pterygium is one in which there has been a corneal ulcer or injury and the healing process has pulled the conjunctiva up on the cornea.

In our opinion, a pterygium seldom stops growing; the growth may be very, very

* Presented at the meeting of the Texas Ophthalmological and Otolaryngological Society, Houston, Texas, December 5, 1947.

slow, but if measured and observed over a period of years, it will be seen that it has progressed.

Surgical treatment

Various procedures have been described for the removal of pterygia. Des Marres was the first to transplant the pterygium after dissecting it from the cornea, McReynolds later modified the Des Marres operation, and his is the procedure most commonly used. We will not go into detail regarding the surgical technique, but we wish to stress several points that seem important to us.

In the first place, the head and cap of the pterygium should be dissected free from the cornea, removing all of the strands and trabeculae on the initial dissection. We generally incise the conjunctiva just adjacent to the body of the pterygium above and below, and then (this is important) see whether the pterygium fits with the least distortion with the head pulled upward or downward, bringing normal conjunctiva over the former site of the growth and adjacent to the limbus. No tension should be exerted.

All the surgical textbooks emphasize that the pterygium should be buried beneath the lower bulbar conjunctiva, but this is not obligatory. It is better surgical judgment to adapt the transplant to its new position in the most normal and natural way, with no tension and no bulging of the body under the conjunctiva.

The head of the growth is anchored beneath the bulbar conjunctiva, either above or below, with a double-armed, black-silk, 6-0 suture. Then the bulbar conjunctival flap is picked up and brought down over the transplanted pterygium and anchored into the body of the pterygium with a single black-silk suture.

Indication for X-ray treatments

During the last two years one of us (H. L. H.) has operated on 36 cases of pterygia. Nine of these had bilateral growths, and both eyes were operated on at the same time. One of the first cases which was operated on soon after resumption of private practice showed a recurrence following the first operation. This patient was operated on a second time, and there was a recurrence within a short time. Since the nasal side of the eye was quite vascular and the pterygium again commenced to extend onto the cornea, the patient was referred for X-ray treatments. Following the first X-ray treatment, the vascularity subsided, and the pterygium stopped growing.

X-ray treatments following surgery are not indicated in every case; in fact, they are contraindicated in the avascular, small, flat type of pterygium. Only the vascular, raised, large type requires X-ray treatment, but these cases need it badly. Of the 36 cases operated on, 10 required X-ray treatment. Our usual procedure is to advise the patients that on the fifth day following surgery, when the stitches are removed, they will be referred to the roentgenologist for one or more X-ray treatments. Our results from the combined procedures have been excellent.

X-ray treatment of pterygium

X rays have been useful in the treatment of various lesions about the eyes. Of these pterygium has been one that has received some attention in the literature. Bucky reported the use of soft X rays in this condition in 1927. Other workers, notably Burnham and Neill, have used Beta rays of radium in the form of radon applied by a special applicator in the treatment of superficial lesions of the eye. Estrada brings out the value of postoperative X-ray therapy in cases prone to recurrence. It has been in these cases that we have used radiation therapy. Choice of the patients to be treated is made by the ophthalmologist after consideration of the patient's history and study of the type of pterygium.

The purpose of X-ray treatment is to help prevent recurrence and growth of the

pterygia. The vascular type, being the kind most likely to recur, is also the type most sensitive to radiation. The effect is produced by the inhibiting action of X rays upon young connective tissue, the epithelial cells, and the blood vessels. This means that the younger the cells of the tissues and the faster the growth, the more pronounced are the effects of radiation. Therefore, we have chosen to treat these pterygia only after surgery, and soon after the operation, while the tissues are in the active reparative phase. In this manner, the maximum effect is produced on the pterygium and also on the scar of the cornea. We begin treatment on about the fifth postoperative day.

Technique of X-ray therapy

The size and shape of the pterygium is measured accurately and a portal is cut out of lead foil which will closely fit the lesion. Then the eye is anesthetized with 1-percent pontocaine, and the lids are held open with a speculum. The lead-foil shield is put in place over the eye and the X-ray beam is directed through the portal (fig. 2). Its direction is angulated to the side of the lesion being treated so that the center of the beam does not go through the center of the orbit.

Unfiltered X rays produced at a voltage of 70 K.V. (H.V.L. 1.0 mm. al.) are used at a distance of 22.5 cm. (9 inches). This gives the longer wave-length, less penetrating rays. The output is 130r per minute so that the exposure time is short and rarely runs over two minutes. Each pterygium is treated in this manner at each treatment, and the treatments are repeated at intervals of 3 to 5 days. It is important to give the treatments in a period of 2 to 3 weeks to take advantage of the altered pathologic processes which follow the operation.

Comment

In this series of cases, 10 patients were given postoperative radiation. Of these two were women. Six of the patients had bilateral lesions and one of these had three pterygia. There was a history of multiple operations and recurrences up to a total of six in one patient.

No statistical analysis of this small series of cases will be attempted. The patients received an average of six treatments, with an average total dosage of 1,100r. Very gratifying results have been obtained. In some of the earlier cases, which have been followed for almost two years, the lesions show no evidence of growth. No complications of any kind have been noticed. We might stress that the tolerance of the cornea to irradiation is much higher than is generally thought so that no adverse effects from this or even higher dosage are to be expected.

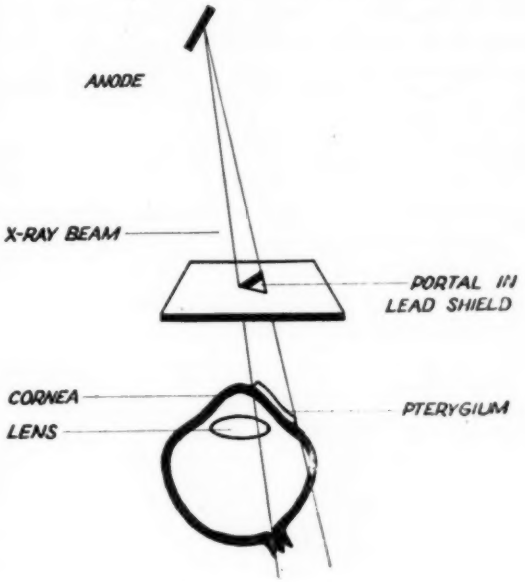

DIAGRAM ILLUSTRATING THE ANGLED X-RAY BEAM STRIKING THE PTERYGIUM.

Fig. 2 (Hilgartner, Wilson, and Wilson). Diagram illustrating the angled X-ray beam striking the pterygium.

We are presenting this combined method of treatment of pterygium because of its simplicity, availability, and its effectiveness in stopping the growth and preventing the recurrence of these lesions.

202 West 13th Street.
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CAPILLARY FRAGILITY AND CUTANEOUS LYMPHATIC FLOW IN RELATION TO SYSTEMIC AND RETINAL VASCULAR MANIFESTATIONS: RUTIN THERAPY*

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With increasing frequency it is recognized that vascular accidents, particularly of arterioles and capillaries, are not solely the result of simple elevation of blood pressure with mechanical rupture, or of degenerative atheromatous changes, such as may lead in larger vessels to massive hemorrhage or dissection of the wall, but are due to some defect in the architecture of the vessel, similar to but not identical with the disorders seen in scurvy, occurring in many persons with normal blood pressures, and bearing no relationship to the actual level of pressure in hypertensives.

Thus, we have long pondered over the cause of the ecchymoses and bruises so frequently seen in a certain type of woman—usually blonde, well nourished or obese, with skin of fine texture, good complexion —whose arms and legs are covered with numerous black and blue spots which result from the slightest pressure or injury. These bruises are not due to scurvy, are not relieved by enormous quantities of ascorbic acid, and appear in patients without other ascertainable cause.

Working in Europe in 1936,[1] Szent-Györgi isolated from the rinds of citrus fruits a substance which corrected or prevented artificially produced capillary fragility in experimental animals and for which he proposed the name citrin, briefly referred to as vitamin P (for permeability). Since it is not a vitamin, none of the effects that it remedies having been observed in dietary restrictions, and since its action is more concerned with capillary fragility, the designation of vitamin P is obviously inappropriate.

Citrin is composed of at least two distinct glucosides, hesperidin and eriodictyol, the former of which varies greatly in physiologic activity while the latter is inert. Presumably much of the activity of this impure mixture is due to rutin,† derived at present from the flowering leaves of immature buckwheat. Rutin is a flavonal glucoside, and is now available in an accurate and stable form.

The pioneer work on capillary fragility and its control has been done in this country by Griffith, Lindhauer, Couch, and Shanno,[2-6] and we have drawn freely from their results, especially as exhibited at the Scientific Assembly of the American Medical Association, July, 1946, to supplement the work which we are reporting. Fragility is measured by one of various techniques, all of which depend on the number of petechiae produced in a measured area following application of a tourniquet, commonly a blood pressure cuff, for a definite time and pressure. Griffith and his associates prefer the Göthlin technique (see references), while we have used a modification of the Wright and Lilienfeld method, performed in the following manner.

A circle 2.5 cm. in diameter is outlined in the antecubital fossa, the upper edge lying 4-cm. below the crease of the elbow, and all confusing blemishes in this area are marked off. The cuff of the blood-pressure manometer is then applied in the usual position, the pressure set at a point midway between the previously determined systolic and diastolic pressure. After five minutes the pressure is released, and one-half hour later the petechiae in the circle are counted. On the average, normal subjects of varying

* Presented in part before the Chicago Ophthalmological Society December 16, 1946. From the Presbyterian Hospital, Chicago.

† Supplied for this work by Abbott & Co., Chicago.

ages will exhibit not more than 10 petechiae after this procedure.

Our reason for selecting this method rather than that of Göthlin (which elevates the pressure to 35 mm. Hg for 15 minutes, followed in an hour by pressure of 50 mm. Hg for 15 minutes) is that with the great variations encountered in both systolic and diastolic pressures, determination of the number of petechiae in a given individual with a certain blood pressure will more nearly represent the state of capillary fragility than a more arbitrary procedure. We have as yet found no reason to modify this technique, but the matter is open to debate and correction. However, it is clear that any such procedure, if carefully standardized, will give accurate and reliable results in the hands of those employing it.

The first comprehensive work of Griffith and Lindauer[2] may be summarized as follows. Increased capillary fragility, occurring in approximately 20 percent of 1,600 hypertensives, irrespective of the blood-pressure level, is accompanied by an incidence of cerebral or retinal hemorrhages and death, varying from 6 to 10 times that found in hypertensives of comparable levels showing normal fragility. This abnormal fragility can be restored to normal by rutin therapy in 75 percent of cases, becomes borderline in 15 percent, becomes normal but relapses in 4 percent, and remains abnormal despite large dosage in 6 percent.

Furthermore, restoration of normal fragility is followed by a decrease of vascular complications to the approximate frequency encountered in hypertensives with normal fragility, only to be followed by a return of greater incidence in relapse due to discontinuance of therapy or failure of treatment.

Finally, thiocyanate therapy, extensively used for control of hypertension, produces in a significantly large number of cases an increased fragility, thus predisposing the patient to the hazard of vascular complications that are frequently more serious than uncomplicated hypertension, which tendency is fortunately controlled by rutin.

These findings of Griffith and Lindauer are in complete accord with our findings in a smaller series of hypertensives and are in marked contrast to the results obtained in diabetics, to be discussed later.

The determination of the rate or expectancy of vascular complications in normal and increased fragility is obviously difficult, since one cannot select two groups of 100 cases each, with 20 exhibiting normal fragility, proceed to treat one group with rutin, and conclude either that, in the treated group, this or that specific individual would or would not have met with some hemorrhagic phenomenon; or that, in the untreated cases, any specific complication would have been prevented by therapy.

Consequently, it is necessary to adopt a post-hoc method, whereby a comparison of the incidence of accidents is made after they have occurred, and then related to the state of fragility. To complicate the situation further, we cannot withhold treatment, or withdraw it, in order to obtain additional data on human beings, thus exposing them to the danger of disaster.

Rutin, available in 20-mg. and 60-mg tablets, has conventionally been administered in doses of 80 to 100 mg. daily. However, in resistant cases, or where rapidity of action is desirable, 240, 300, or even 500 mg. daily may be given for long periods without any evidence of untoward or toxic effects. When apparently adequate dosage has been established, the tourniquet test should be done every 4 to 6 weeks to forestall relapses due to possible increase of rutin requirement, bearing in mind that the result is not valid if done within three weeks of a previous test on the same arm.

During an acute shortage of rutin while these studies were in progress, we fortified our meager dosage with large amounts of ascorbic acid, such as 1,000 mg. intravenously once or twice daily, or 200 to 300 mg. orally if tolerated. Although the struc-

tural formula of rutin, a flavonal glucoside, in no way resembles that of ascorbic acid, their action in capillary bleeding is similar, and we found that this addition so enhanced the action of rutin, that administration of 40 to 60 mg., by itself ineffective, resulted in satisfactory and prolonged control of increased fragility. Recent work demonstrating control by Tocopherol of hemorrhage in dogs produced by large doses of Stilbestrol, led to a trial of this substance in a similar manner, but in dosage of 300 to 400 mg., daily. Tocopherol* failed to modify or enhance the action of rutin.

Cutaneous lymphatic flow (C.L.F.),[5] when increased, indicates in many instances increased permeability of vessel walls and was found to accompany increased fragility in about one third of the cases.

Apart from increased permeability, increased C.L.F. occurs in increased capillary pressure due to renal or pituitary disease, is decreased in congestive heart failure, lymphatic block (such as in phlebitis accompanied by peri-venous inflammation), and in edema of nephritis and nephrosis. Work in progress indicates marked reduction or absence of flow in edematous lower extremities of nephrotic patients, while remaining normal in the noninvolved areas of the arms, increasing to normal values in the legs as edema subsides.

As one example, a girl with pure nephrosis or nephrotic syndrome (total serum proteins 2.63, albumin 1.01, globulin 1.62), persistently four-plus albumin, and massive edema of all tissues below the diaphragm, had normal capillary fragility in both antecubital and popliteal spaces, with C.L.F. normal in arms and absent in her legs. During five days she lost 40 pounds as the result of strict electrolyte management, administration of two units of plasma daily for the preceding seven days (this was discontinued at the onset of diuresis), and maximum doses of vitamin K. C.L.F. became normal in the legs (1 to 6 cm. in 14 minutes) in spite of the fact that there was no decrease in the quantitative 24-hour output of protein.

This indicated either that (1) a return to normal C.L.F. was not paralleled by improved capillary permeability, at least in so far as the kidneys were concerned; (2) the decreased C.L.F. in the edematous portions was due to mechanical forces rather than derangement of permeability, which in this instance would have been decreased; or (3) proteinuria in such cases may be due to faulty protein metabolism rather than changes in permeability.

On the other hand, macular edema, as described in subsequent case reports, responded in some instances with amazing rapidity, paralleling improvement in capillary fragility induced by rutin therapy. Unfortunately, determination of C.L.F. was not available at the time but was presumably increased before therapy. C.L.F. studies are now to be made routinely on such cases. On patients exhibiting increased C.L.F., cyanate therapy is ineffective and may actually be harmful, but, following response to rutin, cyanate may be administered with satisfactory results in those in whom it is ordinarily effective.

In striking contrast to the retinal picture and generally satisfactory response to rutin in the hypertensive and sclerotic patient, is that condition encountered in the diabetic, and more frequently in the patient carrying a moderate degree of hyperglycemia without frank glycosuria.

Briefly, the retinal lesions are small, round rather than flame shaped, are situated deep in the retina, are relatively permanent in size and shape, appearing unchanged in character and location over long periods, and respond little or not at all to therapy.

Moreover, these individuals display a much higher average petechial index than hypertensives and are extremely resistant to rutin, in some cases showing no improvement and in 45 cases, as reported herein,

* Supplied by Hoffman-La Roche Co.

responding with a final average of 29 petechiae following maximum treatment.

In his discussion of edema and hemorrhages of the retina, Duke-Elder[7] states "in most cases the escape of fluid from the circulation is through the capillary walls, and since it is probable that these preserve their permeablity to a great extent so long as the endothelial cells are healthy, the appearance of edema is usually an indication of capillary or tissue damage."

He considers as an essential factor in this process, "an increased transference of fluid to the tissues when the capillary permeability is increased, as in toxic and inflammatory conditions, a process which, when accentuated, leads to the escape of albumin and fibrinogen which coagulate in the form of exudates, and eventually to the escape of the formed elements of the blood as actual hemorrhages."

"It would seem," he further states, "that the essence of retinal hemorrhage is probably capillary dysfunction rather than high blood pressure itself."

Of the many instances of exudation and hemorrhage in the retina, which the ophthalmologist constantly observes, none is more troublesome than that which occurs, in apparently ever-increasing frequency, in diabetes mellitus. Despite almost a century of investigation, the cause and cure of this type of retinopathy still eludes us. Opinion is still divided as to whether the retinal changes observed are specific or are merely the findings which are generally accepted as characteristic of arteriosclerotic or renal retinopathy. Recently the latter view has been challenged by Ballantyne,[8] who found that 50 percent of diabetics with retinopathy exhibited no evidence of arteriosclerosis or hypertension. He contends that the condition is primarily venous in origin and begins with fatty infiltration and swelling of the endothelium of the smaller vessels and proceeds to capillary and venous stasis.

Elwyn[9] also feels that the small retinal hemorrhages observed in diabetes have no relation to the vascular changes of aging, sclerosis, hypertension, and obstruction of veins, but rather are dependent on a prestatic condition in the capillaries of the retina which is in some way related to continuous moderate hyperglycemia.

"This," he states, "is as far as anyone can go unless one would rather assume as a cause of the hemorrhage the presence of toxic substances which cannot be found, or a deficiency of unknown vitamins, which so far have not been discovered."

During the course of the Gifford Lecture before the Chicago Ophthalmological Society in 1946, Friedenwald[10] related that he had observed an increase in the general capillary fragility, as determined by the arm-band test, in patients with diabetes mellitus. Recently Wagener[11] referred to some as-yet-unpublished observations of Foxworthy of the Mayo Clinic. In a group of 85 nondiabetic patients of varying ages and in whom, too, there was no evidence of hypertension, she found an average of 14 petechiae after application of the blood-pressure manometer cuff for 10 minutes; 69 diabetics without retinopathy presented an average of 41 petechiae, and in 44 diabetics with retinopathy, the petechial average was 101.

This paper is a preliminary report of some studies which are being continued and expanded. Until recently rutin was not generally available, and the number of patients treated and the size of the dosage were consequently limited.

In addition to the determination of the capillary fragility other related data were obtained. These include the age, general medical studies, urinalyses, complete blood counts, the bleeding time, platelet count, blood sugar level, blood cholesterol, plasma vitamin-C level, and prothrombin time. The capillary fragility was determined by the positive pressure method described above.

Of the 81 patients studied, 45 had diabetes mellitus, and these diabetics revealed the following pertinent data: their average

age was 55 years, varying from 24 to 73 years; 33 were females and 12 males. General studies revealed complicating mild nephritis in 8, and arterial hypertension in 15. Evidence of arteriosclerosis was present in 13 instances. Incipient or immature cataract was found in 11 patients, and retinopathy was present in 25 of the 45. The average systolic blood pressure was 154 mm. Hg, ranging from 96 to 270 mm., and the average diastolic was 85 mm. Hg, varying from 60 to 130 mm. Glycosuria was present in 11 instances, and blood studies revealed mild anemia in 3, and leukocytosis in the same number. A prolonged bleeding time was found in only one patient and a lowered platelet count in two. The fasting blood sugar ranged between 84 mg. percent and 289 mg. percent and averaged 141. Blood cholesterol was also generally elevated, averaging 320.6 mg. percent. In more than one third of these patients the fasting plasma vitamin-C level was subnormal, averaging 0.74 mg. percent and varying between 0.15 and 1.35. Prothrombin was reduced to 60 percent and 80 percent, respectively, in two instances. Nineteen of these patients took insulin daily.

Capillary fragility averaged 53 petechiae for the whole group and 60 for those with retinopathy. In the 20 diabetic patients without retinopathy the average capillary fragility was 16. Except for three instances the capillary fragility decreased, usually within 12 to 18 days, after the administration of an average daily dosage of 160 mgm. of rutin by mouth, to an average level of 29 petechiae.

Despite the reduction in the general capillary fragility, there was no significant objective improvement in vision or in the appearance of the retinopathy in these 25 patients over periods of 3- to 12-months' observation. However, it may be of great significance that in 18 instances of retinopathy on rutin therapy from 10 to 12 months, there has been no decrease in vision and little or no change in the retinopathy. No effect on the blood pressure, bleeding time, platelet count, and so forth, was observed.

Report of cases

The fundus photos (figs. 1 and 2) will best illustrate the type of lesion with which we were generally dealing.

Case 1. J. D., a white woman, aged 54 years had been a known diabetic for 18 years. Small doses of insulin were employed for the last six months only.

At the time of her original eye examination in December, 1943, a mild degree of retinal angiosclerosis and a fairly marked retinopathy were noted. The fundus picture has remained essentially unchanged and corrected vision, which was recorded as: O.D., 0.1; O.S., 0.3, in 1943, was noted to be: O.D., 0.1; O.S., 0.2−1 in November, 1946.

On May 9, 1946, the blood pressure was 154/84 mm. Hg, and the capillary fragility was 101. A daily dose of 120 mg. of rutin was prescribed. On June 3, 1946, capillary fragility measured 32. Only 10 petechiae were present on testing the capillary fragility on July 23, 1946. After employing no rutin for 10 days the fragility was found to be 44 on September 12, 1946. The last test, on November 7, 1946, after the resumption of rutin therapy, revealed 32 petechiae.

Case 2. A. E., a white woman, aged 54 years, was the private patient of Dr. E. V. L. Brown. She had been a known diabetic for 10 years, under insulin therapy. When first examined in July, 1946, the right fundus presented the picture of an old occlusion of the central retinal artery. There was marked retinopathy of the left eye. Vision was: O.D., perception of light; O.S., 0.2−1, with correction. The blood pressure was 138/74 mm. Hg, and the capillary fragility 18. She was given 120 mg. of rutin daily and on reëxamination on August 19, 1946, the capillary fragility had dropped to zero. When the patient returned on October 17, 1946, she related that she had exhausted

her supply of rutin one month previously. A determination of the capillary fragility revealed 25 petechiae. Rutin was resumed and on November 14, 1946, the fragility was 8 petechiae. At this time vision was: O.D., none; O.S., 0.2, with correction.

In addition to these patients with diabetes mellitus, 36 individuals with a wide variety of ocular and general disturbances have been studied.

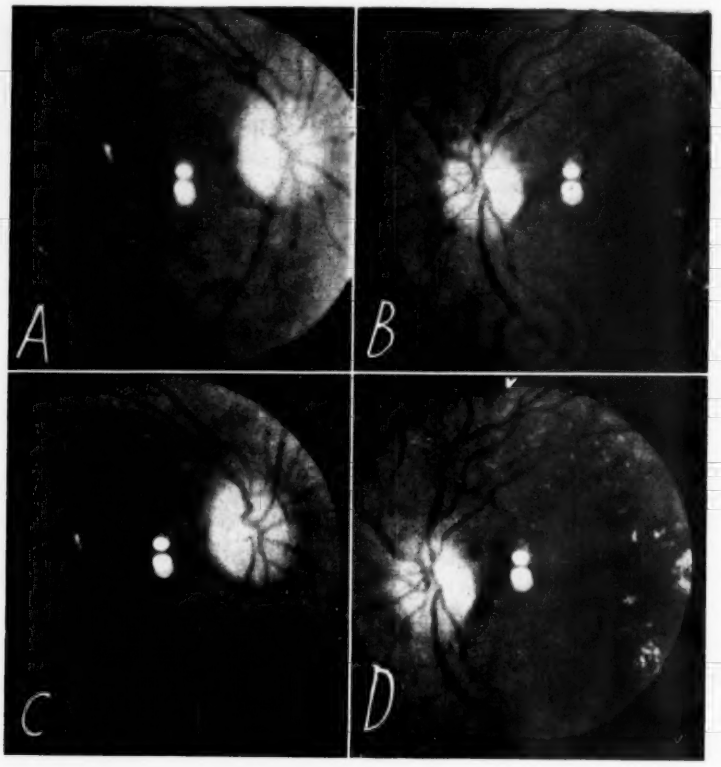

Fig. 1 (Donegan and Thomas). *Case 1.* A white woman, aged 54 years, had been a known diabetic for 18 years. On May 9, 1946, before beginning treatment with rutin (A) fundus of the right eye with capillary fragility equaling 101 and vision, 0.1. (B) Fundus of the left eye at the same time, with capillary fragility equaling 101 and vision, 0.2—1. (C) Fundus of the right eye and (D) fundus of the left eye six months after beginning treatment with rutin. Capillary fragility is 32; vision: right eye, 0.1; left eye, 0.2—1.

Three young men who had experienced recurring hemorrhages into the vitreous, so-called Eales's disease, presented no abnormalities on general physical examination and laboratory studies were negative in two. In these two, capillary fragility was increased to 26 and 32 respectively and, after rutin, has fallen to 16 in each. In the third instance, the prothrombin time was reduced to 60 percent but responded promptly to vitamin K. The capillary fragility of this third patient measured 6 in June, 1946, when he was given rutin, which he has continued, and was reduced to zero when tested three months later. This patient has experienced no recurrence of vitreous hemorrhage during the last six months; whereas during the previous two years bleeding recurred once in six weeks on the average. The period of observation of the other two patients has been limited but one of them has recently experienced a recurrence despite daily doses of rutin of 160 mg.

Of interest are two adults found to have

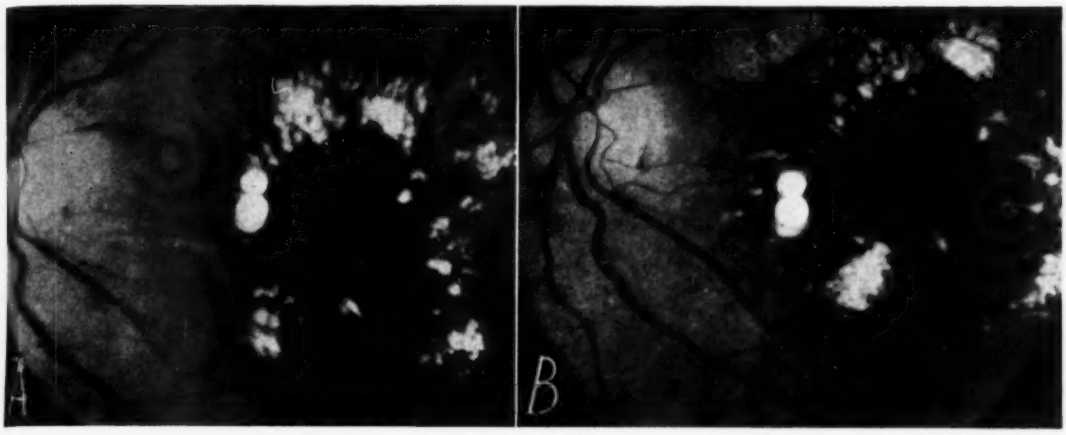

Fig. 2 (Donegan and Thomas). *Case 2.* A white woman, aged 54 years, had been a known diabetic for 10 years. (A) The fundus of the left eye before rutin therapy was begun; capillary fragility is 18; vision is 0.2—1. (B) Fundus of the left eye five months after beginning of treatment with rutin. The capillary fragility is 4; the vision is 0.2.

unilateral macular edema, with corresponding slight reduction in vision and relative central scotomas. The first, a white woman, aged 39 years, with mild vascular hypertension had noted slight blurring of right central vision for six weeks prior to examination. Except for the circumscribed area of edema in the right macula, the fundi and retinal vessels were normal. Her blood pressure was 150/90 mm. Hg, and the capillary fragility measured 81 on May 9, 1946. Rutin (160 mg.) daily was prescribed and seven days later she reported that the vision had cleared completely. Examination revealed only a few minute, yellow dots in the right macular region. No central scotoma could be elicited and the petechial index was 16.

In the second case, that of a 38-year-old white man, the history was similar and examination revealed a circumscribed area of edema in the left macula, with a corresponding relative central scotoma. General physical examination was essentially negative and the blood pressure was 112/72 mm. Hg. Only two petechiae were found on testing the fragility but 160 mg. of rutin were prescribed. Two weeks later he reported that vision in the left eye had returned to normal, and reëxamination revealed no central scotoma. The left macula appeared normal except for three tiny yellow dots. At this time the capillary fragility was zero.

Two of three patients with high myopia and macular hemorrhages had normal capillary fragility, while one presented 63 petechiae after application of the cuff pressure. The fragility decreased to 16 after rutin therapy but no significant improvement in vision or the fundus picture was noted in this patient nor in the other two.

No improvement in vision nor in the ap-

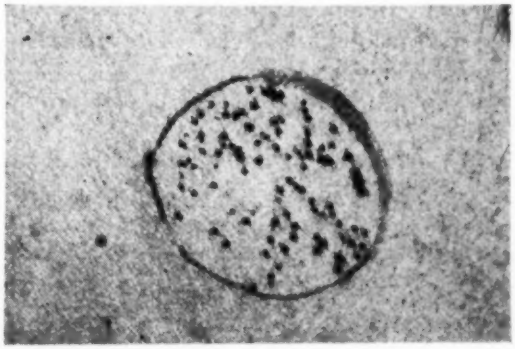

Fig. 3 (Donegan and Thomas). Demonstrating an abnormal number of petechiae in a 2.5-cm. circle in the antecubital fossa after application of the blood-pressure cuff for five minutes at a pressure midway between the previously determined systolic and diastolic pressure.

pearance of the retina was found after rutin therapy in two cases of occlusion of branches of the central retinal vein.

However, one patient, a 72-year-old white woman, suffering from mild generalized arteriosclerosis and hypertension experienced a sudden loss of left vision which was attributed to occlusion of one of the disc vessels. A remarkable recovery of vision and coincident clearing of the edema and petechial hemorrhages took place under rutin therapy, while the capillary fragility dropped from 46 to zero.

No increase in general capillary fragility was demonstrable in three individuals with central choroiditis or in two patients with disciform degeneration of the macula, and no improvement in vision occurred after administration of rutin.

The remaining 20 patients studied were hypertensive and arteriosclerotic individuals with a heterogenous collection of retinal complications. Capillary fragility was abnormally increased in all of these and was generally reduced after the administration of rutin. In numerous instances favorable changes in the retinal complications were observed after institution of rutin therapy.

In summary, it would seem that our findings are similar to those of Friedenwald and Foxworthy, who recently found the capillary fragility generally increased in diabetes mellitus, and even more markedly increased in diabetic patients with retinopathies. An increased capillary fragility in vascular hypertension, as reported by Griffith and Lindauer and by Shanno, was also demonstrated in our small series. The general capillary fragility was almost universally reduced by the ingestion of rutin in 51 patients.

Although no significant improvement in vision or decrease in the extent of retinopathy was observed in 25 patients with diabetes mellitus, we feel that the subject warrants further study. The retinal lesions in diabetes mellitus are noted for their chronicity and inertness, and in the patients included in this study the retinopathy was marked and undoubtedly irreversible.

Conclusions

1. Capillary fragility was found to be abnormally increased in arteriosclerosis and hypertension, to a greater extent in diabetes mellitus, and most markedly in diabetics with retinopathies.

2. Rutin has been found to decrease capillary fragility, but not to normal levels in diabetics with retinopathies.

3. Although no significant improvement in vision or decrease in retinopathy was observed in diabetes after rutin therapy, it may be significant that no loss of vision or increase in retinopathy occurred during a period of 10 to 12 months' treatment.

4. Rutin appears to be of benefit in the treatment of Eales's disease and central serous retinopathy.

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MILD GLAUCOMA*

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The classification of glaucoma into acute, chronic congestive, and chronic simple forms is based on the clinical course of average typical cases. It does not take into account, however, the degree of severity of the disease. The division of the cases into narrow- and wide-angle types suffers from a similar inadequacy.

To the patient and his family, it is more important to know the prognosis of his disease than to receive an accurate diagnosis. In a series of 373 cases of primary glaucoma,† we observed 40 patients in whom the disease remained unusually mild throughout the entire course. Several of these patients were observed for many years. Some of the cases were discovered accidentally on routine examination, others were diagnosed because of the presence of glaucoma in other members of the family, while still others complained of mild symptoms of glaucoma.

In these patients the ocular hypertension is not very high, and generally is well tolerated. A good response to miotic therapy is the rule. The fields show no deterioration during the entire period of observation, and the fundi and vision remain normal.

Those cases in which the elevation of tension was the only sign of glaucoma were included if a provocative test or pupillography, or both, gave a positive result. Brief histories of 16 of these cases are reported in this paper. In addition to these, there are several patients in our series who showed mild glaucoma in one eye, and a severe or moderately severe form in the other. One such case (Case 17) is included in this paper.

CASE REPORTS

Case 1. Lottie C., aged 60 years, was told 25 years ago by an eminent ophthalmologist that she had glaucoma. She has had recurring episodes of ocular discomfort and blurring of vision. Tonometric readings have varied between 20 mm.‡ and 35 mm. in both eyes. The Knapp adrenalin test was positive in 1922 but homatropine failed to elevate the tension. Her anterior chambers and angles are intermediate in depth. She is a moderate hyperope. Her visual acuity has remained 20/20 in the right eye and 20/25 in the left eye over the past 18 years of observation. Visual fields and fundi are normal. The pupillographic curve is similar to that found in primary glaucoma. Pilocarpine (½ to 1 percent) relieves symptoms. Her brother and father suffered from glaucoma.

Case 2. Samuel D., a 64-year-old lawyer, was first seen in 1927 when he complained of intermittent pain in his eyeballs. Tension was normal at that time. In 1934, he saw colored halos around lights on three different occasions. The tension was still normal in both eyes. These symptoms continued at intervals. In addition, there was blurring of vision especially after near work and card playing. The attacks occurred at varying intervals, more commonly in the left eye, and were relieved by 1-percent pilocarpine. The tension was measured frequently over a period of 18 years and was found to be below 30 mm. except on three occasions. In 1939, the tension in the left eye was 33 mm. In 1943 the tension was 36 mm. in the right and 45 mm. in the left. Similar tonometric readings were obtained in 1945. His vision has remained 20/20 in each eye. The left eye is emmetropic while the right

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† Many of these cases were originally patients of the late Dr. Mark J. Schoenberg.

‡ All tonometric measurements were made with the Schiøtz tonometer and readings refer to the 1924 graph according to which 30 mm. Hg represents the upper limit of normal.

eye takes a −0.50D. cyl. ax. 180°. Fundi and fields are normal. His anterior chambers are deep and angles are wide. Pupillography showed a curve similar to that found in primary glaucoma. Thus it was not until 16 years after the onset of symptoms that a significant elevation of tension was discovered.

Case 3. Samuel F., a 55-year-old physician, began to complain of tearing, blurring of vision, and halos in 1931, three months after a golf-ball injury to the left orbit. The symptoms recurred intermittently for 10 years; yet at no time was the tension found to be elevated even though he made frequent visits to an ophthalmologist. If he used pilocarpine before a movie, he could prevent the subsequent appearance of halos. Between March, 1942, and October, 1943, the tension of his left eye was found to be 36 mm. on four different occasions. His right eye showed an elevation to 33 mm. on only one occasion. The symptoms were readily controlled by pilocarpine in dilutions as low as ½ percent. He has been suffering from left-sided familial migraine since 1913. The chamber angles are narrow. Vision of the right eye is 20/20 with a +4.0D. sph. ⊂ +2.5D. cyl. ax. 100°; and that of the left eye is 20/20 with a +3.5D. sph. ⊂ +2.75D. cyl. ax. 70°. Fundi and fields have remained normal. Pupillography shows curves similar to that found in primary glaucoma.

Case 4. Sarah A., aged 63 years, was first seen in 1937 when she complained of occasional blurring of vision. Examination revealed bilateral incipient cataracts. The fundi were normal and the tension was 26 mm. in each eye. The vision of the right eye was 20/20 with a +0.50D. sph.; and of the left eye, 20/30 with a +0.25D. sph. ⊂ +0.25D. cyl. ax. 180°. She was advised to use scopolamine (1/5 percent) three times a week in each eye. For nine months, while using the scopolamine, she was seen on several occasions and her tension was always below 26 mm. Hg (Schiøtz). She stopped medication and had no subjective symptoms, except occasional blurring of vision, for the next eight years. In May, 1945, in the course of a routine examination, her tension was 33 mm. in the right and 31 mm. in the left eye. One month later, it was 49 mm. in each eye, but dropped to 28 mm. one hour after the instillation of 2-percent pilocarpine. At this time, the patient was going through emotional stress. Pilocarpine (1 percent), used twice daily, has controlled the tension to date. Chamber angles are wide. Fundi and fields have remained normal. This patient has nerve deafness and obesity. She is one of six siblings of a consanguinous marriage (first cousins). There are no other cases of glaucoma in the family. However, one brother, her mother, maternal grandmother, and several other members of her mother's family had defective hearing. Her mother also had diabetes.

Case 5. Carrie K., a 62-year-old musician, was first seen in 1929 on routine examination. Her vision was 20/20 in each eye with +0.50D. sph. Tension and fundi were normal. She was seen at regular intervals, and it was not until August, 1944, that the tension in her left eye was found to be elevated to 33 mm. She was not given any medication. At the next visit, one month later, the tension was 40 mm. in the left eye and 28 mm. in the right. She was placed on 1-percent pilocarpine, two times a day. Her tension has been below 30 mm. up to the present time. Vision, fundi, and fields have remained normal up to the present. Pupillography shows a curve similar to that found in primary glaucoma. The patient does not complain of any ocular symptoms, except for tearing of the right eye which is due to partial obstruction of the lacrimal duct. She is a highly sensitive and a high-strung individual.

Case 6. Fay L., a 55-year-old housewife, was first seen in September, 1942. For two years previously, she suffered from eye-strain and sensitivity to light. She would fatigue easily and could not do her work.

A diagnosis of glaucoma was made at a hospital clinic in February, 1942. When she first saw Dr. Schoenberg, she was using miotics three times a day in each eye. She was also being treated for early menopausal symptoms and obesity. Vision was 20/20 in each eye with +0.25D. cyl. ax. 180°. Tension was maintained below 30 mm. in both eyes with 1-percent pilocarpine, three times a day. On one occasion (November, 1943) the tension in the left eye was 41 mm. and in the right 35 mm. Vision, fundi, and fields have remained normal. Pupillography, in 1942, showed a curve which contained elements similar to those found in glaucoma in addition to features suggestive of other neurologic disturbance.

Case 7. Suzanne M., a 66-year-old housewife, has been under observation since 1925. She has never had any ocular complaints. In June, 1945, routine tonometry gave a reading of 39 mm. in each eye. No treatment was started because she was advised to have a pupillographic examination. The patient left town and reported again in December when the tension was found to be R.E., 35 mm. and L.E., 41 mm. She went south and on her return visit in March, 1946, the tension was 36 mm. in each eye. Pupillography at this time revealed a curve which combined the usual pupillographic findings in cases of glaucoma with those of essential hypertension. She was then placed on 1-percent pilocarpine, twice a day. Her tension now fluctuates between 26 mm. and 33 mm. No symptoms referable to glaucoma have been experienced. Fundi and visual fields have remained normal up to the present. Vision is 20/20 in each eye with +0.50D. cyl. ax. 180° for the right eye; and +0.50D. sph. for the left. Gonioscopy shows wide angles. She developed diabetes in 1944 and also suffers from mild essential hypertension.

Case 8. Lillian R., a school teacher, aged 60 years, has been followed since 1931. She had no ocular symptoms until 1941 when she complained of eye fatigue after reading. Routine tonometry revealed tension of 28 mm. in each eye. In 1943, the tension was 36 mm. in each eye. Pilocarpine (½ percent) was prescribed but patient used it very irregularly. In 1945, the tension was again 36 mm. in the right eye and 34 mm. in the left and has remained at about 30 mm. Fundi show shallow cupping. Vision has remained 20/20 with +2.50D. sph. on each eye. Fields have remained normal. The patient also suffers from essential hypertension, diabetes, and obesity. Her mother, four sisters, and two brothers have diabetes. She also suffers from nerve deafness of the left ear.

Case 9. Carrie N. was first seen in 1921 at the age of 35 years. In 1924, she had occasional blurring of vision and ocular discomfort in the left eye, especially in bad weather. The tension was normal at this time. The Knapp adrenalin test resulted in mydriasis, generalized vasoconstriction, and hysterical crying. The jugular compression test was positive in the left eye but failed to raise the tension in the right eye. Homatropine failed to raise the tension; however, pilocarpine lowered tension from 26 mm. down to 14 mm. Elevation of tension was first noted in the left eye in 1928. Although the tension was normalized by pilocarpine, the symptoms persisted. Iridencleisis on the left eye, in 1941, controlled both symptoms and tension. The right eye remained normal until 1944 when the ten sion was 36 mm. Vision has remained 20/15, O.U. She has a moderate degree of hyperopia. Anterior chambers are shallow. Fundi and visual fields have remained normal. She suffers from asthma and essential hypertension. The pupillographic curve was similar to that found in primary glaucoma. Her mother, sister, and several other members of the family suffer from glaucoma.

Case 10. Rosa L., a sister of Carrie N. (Case 9), was first seen in 1921 at the age of 45 years. Because of her symptoms of recurrent blurring of vision of the right eye and a history of glaucoma in her mother,

she was carefully watched for glaucoma. Except for a positive reaction to the Knapp adrenalin test, there was no clinical evidence of glaucoma until 1927 when an elevation of tension to 42 mm. was noted in the right eye and to 36 mm. in the left. Miotics controlled the tension. Homatropine and ephedrine used as provocatives failed to raise tension significantly. Vision has remained 20/20 in each eye with −3.0D. sph., O.U. The fundi and fields were still normal when last examined in October, 1946.

Case 11. Gussie F., a 64-year-old woman, had an attack of acute glaucoma in 1932 at the age of 49 years. The attack subsided under miotics, but eight months later she suffered from another episode of congestion in the left eye. In 1933, she had another attack. This one responded to miotics, but she continued to have frequent elevations of tension with mild congestive symptoms until 1942 when a severe congestive attack necessitated iridencleisis. Tension in the right eye remained normal until 1945 when it was found to be 36 mm. on two separate occasions. She had no symptoms in this eye. Vision has remained 20/20 with +1.5D. cyl. ax. 90°, O.U. Fields and fundi have remained normal. Her sister has glaucoma.

Case 12. Norma P., aged 49 years, began to see colored halos in 1940. In 1941 she was refracted by another ophthalmologist who told her that her eyes were normal. She came under the observation of Dr. Schoenberg, in 1943. In view of the symptoms and the fact that her sister (Gussie F., Case 11) suffered from mild glaucoma, the patient was kept under careful observation. Nevertheless, a rise of tension was not observed until five months later. At this time the tonometric reading of the left eye was 43 mm. Under pilocarpine the tension dropped to 25 mm. on the following day. Pupillography showed a curve in both eyes similar to that found in glaucoma. The right eye has remained normal. Vision is: R.E., 20/25 with a +2.0D. sph. ⊂ +1.0D. cyl. ax. 160°; L.E., 20/20 with a +1.25D. sph. ⊂ +1.5D. cyl. ax. 180°. Fields and fundi have remained unchanged.

Case 13. Celia H., a 64-year-old housewife, began to complain, in 1937, of intermittent colored halos and pain around her right eye, especially after emotional excitement. A diagnosis of glaucoma was made by another ophthalmologist. Since the symptoms were not controlled by miotics, this physician advised surgery. She consulted a second ophthalmologist, but did not tell him her history. He found the tension to be normal and instilled homatropine in her right eye. This was followed by an acute congestive attack in the right eye. She responded to miotic therapy. The left eye has remained normal. Vision is 20/30 in the right eye with a +7.0D. sph. ⊂ +1.0D. cyl. ax. 140°, and 20/30 in the left eye with a +5.5D. sph. ⊂ +0.75D. cyl. ax. 170°. The anterior chambers are shallow and both angles are narrow. Fundi and fields have remained normal.

Case 14. Regina B., a 67-year-old journalist, first noted a heaviness and blurred vision in her right eye, in 1934. The symptoms followed closely upon news of the revolution in Vienna where her family was living. She made her own diagnosis of glaucoma because her cousin suffered from the disease. Tension in her right eye fluctuated between 18 mm. and 33 mm., while that in the left eye measured constantly below 25 mm. Vision has remained 20/20 in her right eye with +4.5D. sph., and 20/70 (amblyopia ex anopsia) in the left eye with +5.0D. sph. Fields and fundi are normal. The course of the glaucoma in this case is very similar to that of her cousin, Celia H. (Case 13).

Case 15. Rachel G., aged 72 years, has been followed since 1934. Aside from mild retinal arteriosclerosis and incipient cataracts, her eyes were found to be normal. In January, 1947, she complained of frontal headaches of one month's duration and occa-

sional blurring of vision. She has slight anisocoria, the right pupil being 4 mm. and the left 3½ mm. Slight cupping of the lower border of the right optic disc was noted, but both discs were normal in color. Vision in the right eye was 20/40 with a +3.5D. sph.; that of the left eye was 20/30 with the same correction. Peripheral fields and blind spots were normal. Tension was R.E., 35 mm.; L.E., 28 mm. Gonioscopy showed wide chamber angles. A lability test showed an increase in the intraocular pressure from 26 mm. to 36 mm. in each eye. Pilocarpine (1 percent) relieved the symptoms and lowered the tension to 22 mm. This case is an example of *early* glaucoma. It is too soon to prognosticate the course which the disease will follow.

Case 16. Ada F., a 65-year-old housewife, had an Elliot trephining operation performed on her left eye in 1926 for an advanced chronic simple glaucoma. At that time, the vision and field had already been greatly reduced. Her right eye remained symptom-free until 1937, when she was seen soon after an attack of cloudy vision which had subsided upon the installation of a miotic. While under continuous miotic therapy for the right eye, tension fluctuated between 16 mm. and 30 mm. until 1944, when a tonometric reading of 36 mm. was noted on one occasion. This patient also has diabetes and thyrotoxicosis. Fundus and field of the right eye have remained normal. Vision in the right eye, when last seen in 1945, was 20/20 with +3.0D. sph. ⌒ +2.0D. cyl. ax. 20°. This case illustrates that the course may be radically different in the two eyes.

Case 17. Osias M., a 70-year-old business man, suffered from a spontaneous retinal detachment of the right eye, in 1920. When first seen, vision in the right eye was 20/30 with +1.5D. sph., and that of the left eye, 20/20 with −1.0D. sph. ⌒ −1.50D. cyl. ax. 45°. Ultimately, he developed a total detachment and complicated cataract in the right eye. He was examined at frequent intervals and the ocular tension was always within normal limits. In 1935, the patient complained of discomfort and ocular fatigability in his left eye after close work. Even on this occasion the tension was R.E., 23 mm., L.E., 28 mm. However, one hour after the instillation of one drop of 1-percent homatropine, the tension of the left eye rose to 39 mm. He used ½-percent pilocarpine two times daily and the tension of the left eye fluctuated between 22 mm. and 33 mm. On another occasion, in 1942, 1-percent homatropine raised the tension of the left eye from 28 mm. to 39 mm. He developed lens opacities and index myopia. His corrected vision in 1945 was 20/70, and his fundus showed shallow cupping. Although no pallor of the disc was noted, a slight degree might have been masked by the presence of nuclear sclerosis. The peripheral field remained normal and the central field showed a small Bjerrum scotoma in 1945, 10 years after the diagnosis of glaucoma was made.

Comments

Mild and atypical forms of glaucoma offer a challenge in diagnosis. Many ophthalmologists have, at various times, emphasized the importance of recognizing the disease when signs and symptoms are absent or minimal. Several terms have thus found their way into the nomenclature. Among these are "preglaucoma"[1] and "prodromal glaucoma."[2] These terms are generally used to describe not the course of the disease but a specific stage in its life cycle. The choice of the terms is unfortunate since they imply that glaucoma has not yet become clinically manifest. It is an established fact that any disease may occur in a mild form and remain mild throughout its entire course. Jacobson[3] mentions a case of "prodromal glaucoma" of 28 years' duration. It is obvious that these terms refer, in reality, to clinical glaucoma which is either early or mild, or both.

An example of true preclinical glaucoma

is the normal eye in a monocular case. Five cases in our series showed a time difference of 8 to 15 years in the onset of glaucoma in the two eyes. In some instances provocative tests such as the Schoenberg[4] jugular compression test were negative in the normal eye. However, in all monocular cases pupillography showed a similar curve for both eyes.[5]

Another example of preclinical glaucoma is the glaucoma-free interval in the acute and chronic congestive forms. In several cases there was a time interval of more than five years between congestive attacks in the same eye. In one case, the left eye remained apparently free from glaucoma following an Elliot trephining operation until 18 years later, when a congestive attack occurred in the same eye.[6] Provocative tests may be negative during this interval. The glaucoma-free intervals should not be confused with the stage which follows a congestive attack, when the tension is often lower than in the other eye. One of our patients who complained of recurring attacks was considered a hypochondriac for one year by several ophthalmologists, because they always found the tension to be low normal. The subsequent course proved that he had chronic congestive glaucoma. If due credence is given to the patient's subjective symptoms, a diagnosis of glaucoma will be made earlier.

The best way to study the preclinical phase of glaucoma is to utilize normal individuals. Since the incidence of the disease in glaucoma families is greater than in the general population, a study of the preclinical stage of glaucoma may be facilitated by limiting one's self to the unaffected members of these families,

In a study of 30 such families from a genetic point of view, it was found that certain individuals, while themselves clinically normal, transmit the disease to their offspring. Since the inheritance is of the dominant type, one of the normal parents must of necessity carry the gene for glaucoma and may therefore be considered to have "genetic glaucoma." Thus genetic glaucoma is one type of preclinical glaucoma.

Pupillographic studies are now being carried out to discover whether pupillography may be an aid in the recognition of such preclinical cases. Three out of 14 children of glaucoma parents showed a curve similar to that found in primary glaucoma. In the general population, the finding of such a curve is extremely rare.

It is obvious that the terms "preglaucoma" or "prodromal glaucoma" should not be used in referring to the clinically established disease. The terms "early glaucoma" and "mild glaucoma," however, are descriptive of the clinical behavior of the disease. It is true that, in the early stage, it is not always possible to predict the future course which the disease may take. The case may be regarded as an early glaucoma until the physician is relatively certain that it is going to continue as a mild case. Of course, a certain number of these cases may later become more severe.

The term "mild glaucoma" was deliberately chosen because in trying to classify these cases in the conventional manner, we found it impossible to evaluate the symptomatology. If patients have symptoms of occasional bluring of vision, slight ocular discomfort, tearing, occasional halos, and difficulty in reading, are we justified in labelling them as chronic congestive glaucoma, even though congestion of the eye is never observed either by the patient or by the physician? There are other cases which have no symptoms nor loss of field and yet show fluctuations in tension. They cannot be unqualifiedly placed in the same category as a typical case of chronic simple glaucoma. In mild glaucoma conventional classifications break down and one has to identify the case according to its clinical behavior.

The diagnosis of glaucoma in such cases is often extremely difficult. Tonometric measurements form part of the routine examination in this office in all patients over

40 years of age. Tensions of 30 mm. or over are considered suspicious and warrant further investigations by means of provocative tests and pupillography. Of course, the reliability of the tonometric readings depends on the proper technique, care of the instrument, and accurate calibration. More important, however, than the numerical reading, is a difference of tension in the two eyes. We agree with Downey[7] that a difference of 4 mm. between the two eyes is significant; wide fluctuations in tension over a period of time are very suggestive even if the tension never exceeds 30 mm.

Among the provocative tests, we consider the lability test[8] and the water-drinking test[9] as the most reliable ones. A history of glaucoma in the family should make one less ready to dismiss the patient's symptoms even in the absence of objective signs. Such patients form excellent material for the study of the earliest phases of the disease. Pupillography has proved a useful adjunct in the diagnosis of a doubtful case.

If a case appears to have a mild form of glaucoma, it is not necessary to rush in with miotic therapy. It may be of benefit to the patient, and it certainly is to the advantage of the physician, to observe the case without masking its course by treatment. Placebos may be useful for psychologic reasons. Many patients are labelled as glaucomatous on the basis of one abnormal tension reading and are told that they must use drops for the rest of their lives.

Even if a patient is known to have mild glaucoma, it may be advisable for him to abstain from miotics as long as the symptoms are minimal and the fields show no signs of deterioration. It is worth mentioning that in some patients, the symptoms are even aggravated by the use of pilocarpine or other miotics. Since the earliest changes in the field occur in relation to the angioscotomas, it goes without saying that the fields should be carefully plotted on a tangent screen or stereocampimeter with small test objects and under standard conditions. We are using a tangent screen without markings from which the field is transferred by means of a pantograph.[10] (See this issue of the JOURNAL page 728.)

As in all forms of glaucoma, one can learn a great deal about these mild cases through long observation. The cases presented have been chosen with this in mind. For example, some patients tolerate moderately elevated tension for relatively long periods of time without showing any symptoms, ophthalmoscopic signs, or loss of field. We have observed such instances even among the more severe forms of glaucoma. Can ocular hypertension within certain limits be regarded as a compensatory mechanism which serves a useful purpose in maintaining the normal function of the eye? This point has been considered at greater length elsewhere.

It is obvious that surgery has no place in the treatment of a disease which runs such a mild course that none of the visual functions are impaired. Some of the cases, indeed, serve as controlled experiments. Several patients who suffered from glaucoma of equal severity in both eyes had an operation on one eye only; yet the disease followed a similiar course in both eyes. In general, it may be said that any statistical study aimed at evaluating the role of surgery in glaucoma should take into consideration the degree of severity of the disease prior to surgery.

A form of mild glaucoma which was not included in this paper is the syndrome of recurrent glaucomato-cyclitic crises.[11] This is a form of recurring attacks of unilateral glaucoma with mild cyclitic signs. It is probably a clinical entity and may be regarded as intermediate between primary and secondary glaucoma.

We have already referred to the fact that in many instances one eye ran a severe course while the other eye followed a mild course. Even among glaucoma patients in general, the two eyes may occasionally vary in the type as well as the severity of the disease.

Our experience with mild glaucoma is not unique. These cases are a part of the everyday practice of ophthalmology. The physician's and patient's attitude toward glaucoma is naturally colored by the dramatic experiences with severe cases. It is well to think of mild glaucoma. It may, at times, prove of great psychologic value for the patient to know that the diagnosis of glaucoma is not incompatible with a good prognosis.

Summary

1. In some cases of glaucoma, the disease is so mild that the fundus, fields, and central vision remain unaltered for many years.

2. Of 373 cases of glaucoma, 40 had the mild form. Seventeen case histories are reported in this paper.

3. Diagnosis is made by prolonged observation, provocative tests, and pupillography. Special attention should be given to the patient's symptoms and hereditary history.

4. Patients suspected of glaucoma should be very carefully studied before a definite diagnosis is made. An attempt should be made, whenever possible, to determine the degree of severity of the disease before instituting miotic therapy.

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A COMPARISON OF ANISEIKONIC TEST INSTRUMENTS AND PROLONGED INDUCTION OF ARTIFICIAL ANISEIKONIA*

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Aniseikonia can be produced artificially in any normal individual by the wearing of afocal glasses which by different lens thicknesses and base curves will magnify the image seen by one eye more than the other. The unequal images produced will interfere with fusion, cause subjective distortion of objects in space when perspective and rectilinear shapes are absent, and if the difference in size is more than approximately 5 percent, fusion and stereopsis are prevented altogether. Lesser differences will cause eyestrain in certain individuals who are unable to maintain suppression under all circumstances. Suppression of parts of an image helps to eliminate the false stereoscopic clues in favor of the more accurate monocular clues in such individuals. Many who have anisometropia or unequal or oblique astigmatic refractive corrections, or have different-sized eyes, have such unequal, yet highly acute, ocular images. Some of these persons will never have comfortable vision until the images are made equal, or one eye is occluded sufficiently to allow continuous suppression.

EXPERIMENTAL PROCEDURE

These experiments involved wearing afocal glasses which magnify in the vertical meridian (axis 180°) of one eye for periods of 25, 37, and 23 days. The final experiment involved oblique aniseikonia, and the lenses were worn for 28 days. Glasses in each instance were worn continuously every waking moment, and measurements were taken twice a day on the test instruments.† The experiments provide evidence about the following questions:

1. How does an individual react subjectively and functionally when suddenly and persistently forced to cope with binocular images which do not match?
2. What is the nature of the subjective and measured adaptation to artificially induced aniseikonia? Will the binocular organization change to promote fusion?
3. How does the space eikonometer[2] correlate with the ophthalmo-eikonometer[3] in measuring artificial aniseikonia? Why is the simple frontal plane alignment[4] unsuitable as a diagnostic test?
4. How sensitive and precise are the two standard instruments? Do they over- or under-estimate the error?
5. What is the effect of homatropine and eserine on the measurement of artificial aniseikonia?

THE AXIS-180° EXPERIMENT

Figure 1 summarizes the first experiment. The subject is the writer, whose eyes are normal except for slight myopia (O.D., −0.75D. sph. ⌒ +0.25D. cyl. ax. 90°; O.S., −0.50D. sph. ⌒ + 0.25D. cyl. ax. 90°) for which he had not been wearing glasses. Aniseikonia, measured without glasses many times before and after the experiment, is: Axis 90°, O.S., 0.4 percent; axis 180°, O.S., 0.8 percent. This means that to equalize the images of the two eyes, the whole left image must be magnified 0.4 percent, and the axis 180°, an additional 0.4 percent. The lines on

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† The writer acknowledges the aid and direction of Kenneth N. Ogle, M.D., and Robert E. Bannon, B.S., of the Dartmouth Eye Institute in these experiments, performed between February 4, 1946, and July 25, 1946. He also thanks Ethel Jean Babbitt, O.D., who was the subject in the experiment of Figure 4. This work differs from that of Herman Burian, M.D., in 1943,[1] in that the size lenses in his experiments were worn at axis 90° instead of axis 180°, and were worn a maximum of 14 days.

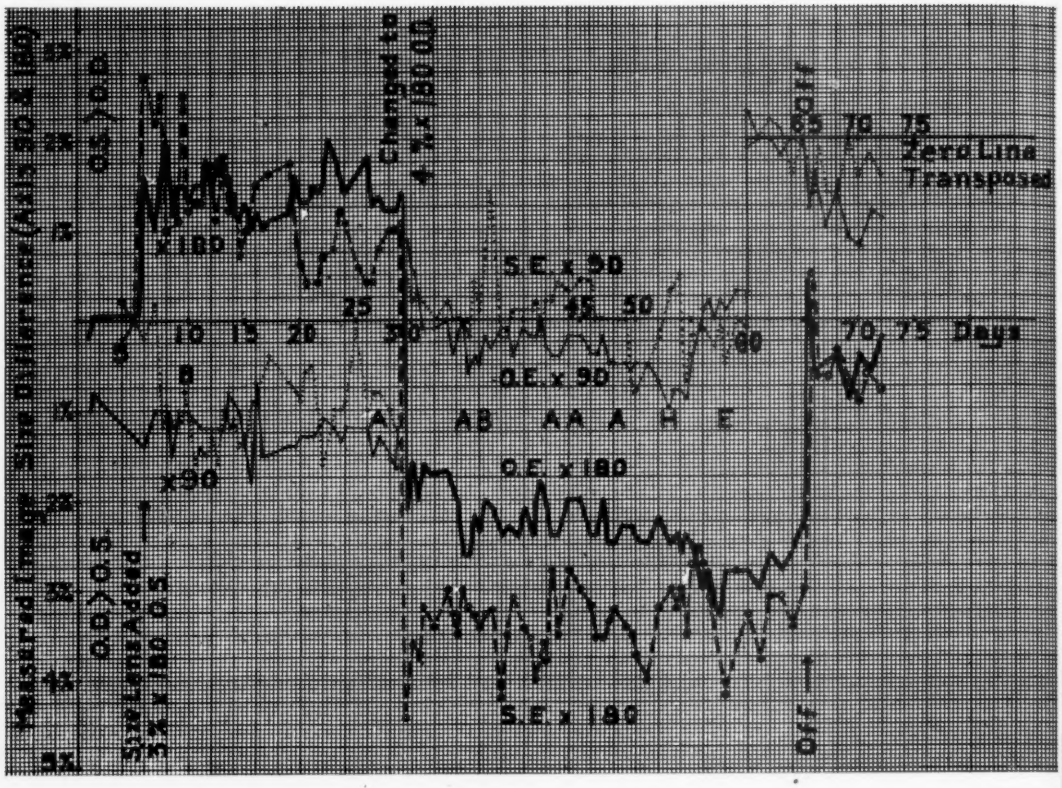

Fig. 1 (Miles). The differences in the size of the images of the two eyes as determined by the ophthalmo-eikonometer (O.E.) at 20 feet and the space eikonometer (S.E.) at 10 feet with the subject (P. W. M.) wearing a meridional size lens of 3.0 percent at axis 180° before the left eye for 25 days, then 4.0 percent at axis 180° before the right eye for 37 days. (A) indicates headache. (B) indicates subjective adaptation to distortion. The heavy lines indicate axis 180° measured, the fine lines, axis 90°.

the graph record this figure: the magnification required over the other eye to equalize the images. The O.S. > O.D. means that under the test conditions, the left image has been found larger than the right in the two meridians the amount specified on the graph.

The graphs may be better understood by referring in Figure 2 to the four diagrams, "P" to "S." "P" shows diagrammatically a stereoscopic card where a tall right image is presented for fusion with a square left image. Obviously, magnification of the left image, axis 180°, is required to equalize the images. Below the abscissa, right images are too large, and above the abscissa left images are too large.

In Figure 1, the abrupt change in the two heavy lines indicates the changes in axis-180° aniseikonia according to the space eikonometer (S.E.) and the ophthalmo-eikonometer (O.E.) when aniseikonic glasses were put on, the fifth day. These glasses were afocal, but the left lens was three times thicker than the right, and was "bent" on the 180° axis so that the upper and lower edges approached the face. This magnified the image seen by the left eye 3.0 percent in the vertical meridian. Neither lens worn blurred the vision perceptibly. Visual acuity at 20 feet was: O.D., 20/25; O.S., 20/20.

Subjective findings with axis-180° aniseikonia

Immediately, there was distortion of surrounding objects, but no particular discomfort. The desk top tipped down toward the right. At dinner the first evening, I dis-

covered an inability to pass a plate of peas. The plate was held 15 degrees off the horizontal. On walking, there was some tendency to try to adjust to the slope visualized, when the feet could much better take care of themselves. A level lawn seemed to slope level. Worse yet, it was impossible to fit a trial frame on a patient's face without a monocular side view. Minor surgery was done without difficulty.

After about five days, "B" in Figure 1, the distortion was no longer annoying in

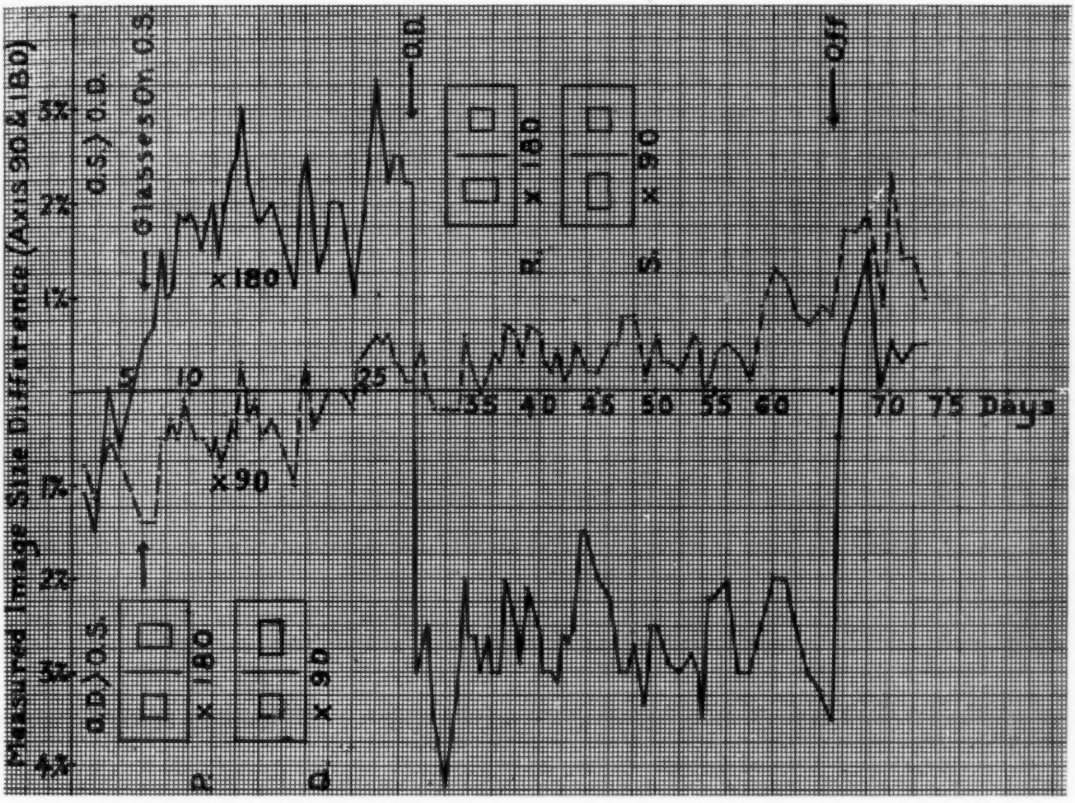

Fig. 2 (Miles). Measurement of axis-90° and axis-180° aniseikonia in the same experiment as in Figure 1, by means of the frontal-plane apparatus (fig. 4). In the axis 180° in subject P. W. M., the test was relatively stable. Axis 90° should remain near the zero line throughout, however. This is the broken line near the abscissa.

up to the left and down to the right for a radius of about 40 feet. More distant lawn was flat, but tipped as it was approached. The left foot appeared smaller than the right and was on a shorter leg. Objects on the right seemed larger and more distant than they were known to be. A wall actually in a frontal plane appeared more distant on the right. Any rectangular object such as a sheet of paper or a magazine appeared trapezoid, larger on the right.

I found it impossible to fix a lampshade ordinary surroundings, but there were occasional mild headaches, points "A." However, when the surroundings contained no perspective or rectilinear detail to increase the probability of mental choice of monocular clues for depth judgments, the distortion returned. For instance, on looking down on a grassy slope, or walking through underbrush, one is forced to depend on binocular clues. A slope was perceived in error as much as 20 degrees. There was a fearful helpless feeling while following a

path through dense underbrush. Branches on the right seemed feet away, but without provocation would approach and strike.

INSTRUMENTS UNDERESTIMATE THE ERROR

In Figure 1, the solid line, axis-180° aniseikonia starts out nearly at zero, but on putting on the glasses on the fifth day, 3-percent axis-180°, left, the image O.S. becomes larger in this meridian than the image O.D. However, the heavy solid line and the heavy dash line (space eikonometer) do not approach a measurement of 3 percent, but fall considerably short. The error was known with certainty, because the glasses worn were tested on the diopto-eikonometer for an eye-wire distance of 9 mm. both at the factory and at the Dartmouth Eye Institute.

The space eikonometer measured correctly the known error at first, but began a progressive undercorrection. The measurement fell in a 25-day period from 2.75 percent to about 0.4 percent, which requires explanation. The space-eikonometer (S.E.) test is based on subjective adjustment of a string target to appear on a frontal plane before the eyes. It is supposedly free of monocular clues, so that judgment is entirely based on stereopsis. Apparently, in this experiment, the adaptation shown was due to some obscure monocular clues which gradually became sure enough to affect final judgment. Stereopsis was given progressively less weight due to memory of its failure in ordinary surroundings.

When the 3-percent lens was removed from the left eye and a 4-percent lens placed axis 180° before the right eye, the adaptation or progressive undercorrection on the S.E. did not take place. In Burian's experiment[1] it was noticed that "the effect of a size lens of 3 percent worn by the two observers H. M. B. and R. E. B. in front of the right eye (10 to 14 days) was reduced by about 50 percent at the end of the experiment, whereas when the lens was worn in front of the left eye, the decrease in effectiveness was only about 20 percent." Burian did not state whether this "adaptation" was limited to readings on the S.E. as in the experiments reported here.

For many years, patients at the Dartmouth Eye Institute have been tested in the "leaf room."[5] This is a box with an open end 6 by 6 feet with a head rest in the center. The box is 7 feet deep, set exactly level with true right angles. The interior is covered with several layers of artificial leaves. Lacking perspective detail and monocular clues, one's judgment of the leaf room depends on stereopsis. One with aniseikonia will state that it is distorted. On placing aniseikonic lenses before the eyes, the end and sides and floor and ceiling all change position. Now, some patients see this distortion with a very weak magnifier before one eye who do not see it when the magnifier is placed before the other. It has been assumed that this is due to dominance. Theoretically, suppression of stereoscopic clues is easier in the recessive eye. These experiments do not confirm this theory. H. M. B. and R. E. B. got adaptation with the lens over the dominant eye; E. J. B. got no adaptation with the lens over the dominant eye. P. W. M. got adaptation with the lens over the recessive but not over the dominant eye.

Moreover, there is no evidence at all of adaptation on the O.E. instrument (solid line fig. 1). This test measures aniseikonia by a comparison of visual angles of about four degrees and requires no use of stereopsis. The adaptation is, therefore, not real. It is due to a change in choice of conflicting monocular and binocular clues in the cerebral centers. Incidentally, monocular clues are more numerous and are given more weight in space judgments than is commonly believed. They include in order of decreasing importance: perspective, overlay, parallax, known size, illumination contrast, position above or below, color, atmospheric haze, and convergence proprioception. Perspective can, under laboratory conditions,

actually predominate over free binocular vision with parallax.

The time factor in aniseikonic tests

In these experiments, perhaps five minutes were spent on the S.E. test, 10 minutes on the O.E., and 25 minutes on the frontal plane apparatus about twice a day. It requires about 10 minutes to get all of the head rest adjustments just right on the O.E., but, once done, the settings can be recorded and duplicated quickly. Adjustment on the S.E. is somewhat simpler. Prof. R. E. Bannon criticized my experiment on the basis that not enough time was taken to measure the full amount of aniseikonia present. He has reiterated for years that one should not be satisfied with the first end point, but should repeat and repeat the test up to an hour or more. Dr. Herman Burian said,[1] "The longer an observer watches a field which is relatively free from perspective factors, the more he frees himself of the memory values of the previous experiences and the more he is apt to give weight to the disparity factors."

The adaptation found in Figure 1 measured on the space eikonometer should not lead to doubts of the instrument's value. The axis-180° adaptation would undoubtedly have disappeared with prolonged testing. Many patients need prolonged testing on this instrument for a different reason. Some, because of aniseikonia or faulty development, have poor sensitivity on the S.E. They do not habitually use stereopsis, and give it no weight in space judgment. Such a patient improves markedly with repeated tests. It is excellent orthoptic training.

Professor Bannon proved his point in the E. J. B. experiment (fig. 3). On the 28th day the measurement had been running along for a week at: O.S., 2.0 percent plus or minus 0.2 percent on the O.E. instrument, when it suddenly went down to 1.6 percent. Her remark that it was adaptation elicited doubt in Professor Bannon. Upon measuring her for an hour, the aniseikonia became 2.6 percent axis 180°, which was more than the 2 percent worn.

Effect of homatropine and eserine

In Figure 1, "H" indicates the use of 2-percent homatropine (3 times daily) in each eye for four days. Plus-2-diopter clip-ons were used for the daily work, but not for measurements. On the O.E., this caused an immediate change in both axis-90° and axis-180° readings, a measurement more nearly the known aniseikonic error by 1 percent in each meridian. Surely homatropine has nothing to do with a memory factor. Immobilization of the lens mechanism or change in the accommodation-convergence ratio may affect the O.E. test. No change was evident in the S.E. test. On the fifth day, eserine ointment was applied, and a myopia of more than 6D. induced. This resulted in a spike not shown on the graph. On the O.E., but not on the S.E., there was more axis 90°, O.D., by 1 percent and less axis 180°, O.S., by 0.75 percent.

Subject E. J. B. found that homatropine for 24 hours did not affect the measurements. She was not undercorrected without it, nor did she show any tendency to adaptation. Her eyes are normal except for myopia: O.D., −2.5D. sph., 20/15; O.S., −2.5D. sph., 20/15. Axis 90° is not recorded in Figure 3, but it ran throughout the experiment (O.S., 0.2 percent plus or minus 0.6 percent on both S.E. and O.E.).

In Figure 1, it can be seen that, while P. W. M. was wearing O.S. axis-180° magnification, the axis-90° reading remained about 1.2 percent, O.D. When the magnification was worn O.D., the axis 90° became about 0.2 percent O.D. This difference is a bit more than experimental error, and is attributed to the induced size effect.[6]

Frontal-plane measurements

Figure 2 shows the aniseikonia as measured on the frontal-plane apparatus. As in Figure 1, the axis-90° line wanders upward, but in a more purposeful way. In

this case, axis 180° is measured quite accurately. The error in axis 90° is evidently due to an increasing amplitude of fusion during the prolonged test, repeated daily for weeks. This can easily be explained by a description of the apparatus and the technique. The accuracy of the method depends on the amplitude of fusion, which is so weak in many patients that the graphic determination of aniseikonia is impossible.

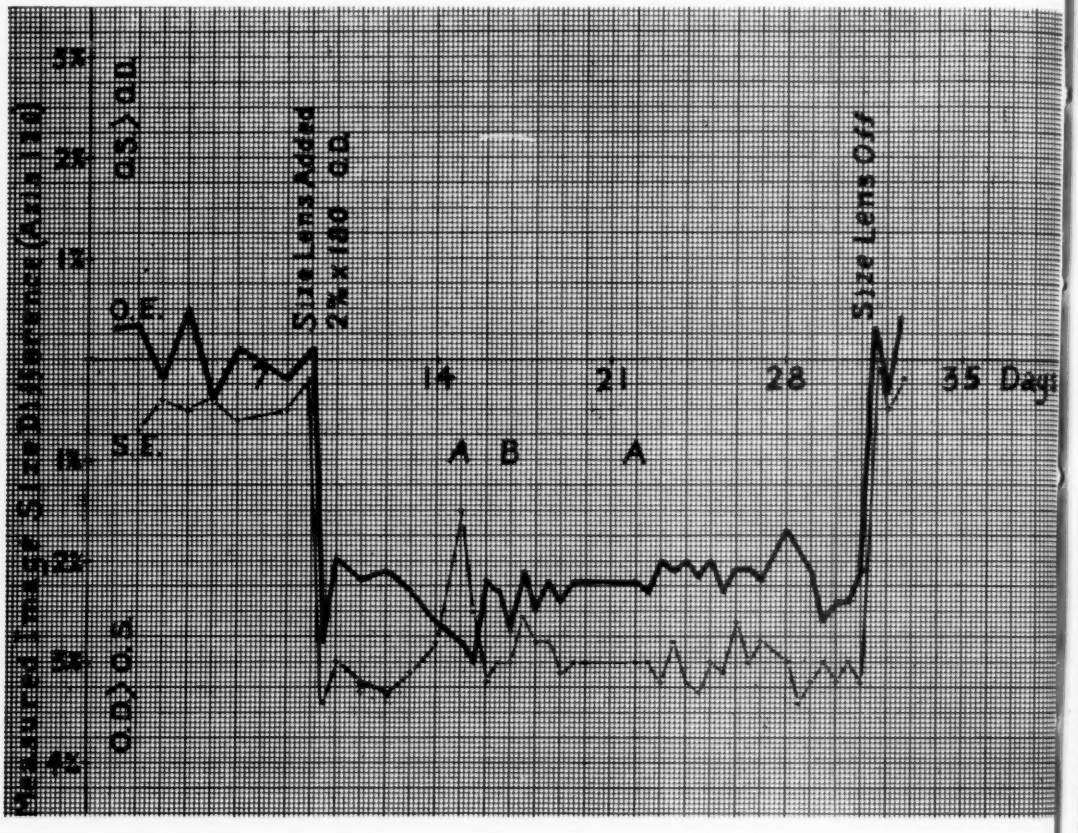

Fig. 3 (Miles). The differences in the size of the images of the two eyes as determined by the ophthalmo-eikonometer (O.E.) at 20 feet and the space eikonometer (S.E.) at 10 feet with the subject, E. J. B. wearing a meridional size lens of 2.0 percent at axis 180° before the right eye. (A) indicates headache (B) indicates subjective adaptation to distortion.

Figure 4 shows the apparatus to consist of a head holder, dissimilar apertures (to prevent their fusion), a target on glass consisting of irregularly shaped and sized spots arranged along a horizontal line level with the two eyes, with a few irregular spots above and below, seen in peripheral vision, which increase the amplitude of fusion. This target is placed before a white background, so that no outside details exist to affect the judgment as to its position. When axis-90° size is placed before one eye, the dots on that side appear larger and are perceived further away. This axis-90° depth change is geometrical and has been explained frequently in the past.[1] The plane is moved by the subject on a vertical central axis until the two sides of the target appear equidistant. The total absence of perspective clues and minimum of monocular clues makes the setting depend chiefly on stereopsis. The number of degrees the plane is rotated proportional to the percentage magnification in the axis-90° direction up to the limit, about 20 percent

When axis-180° size is placed before one eye, it has exactly the opposite effect as axis 90°, with equal but opposite rotation of the target plane up to about 6 to 8 percent. This is the induced size effect of Ogle.[6] Stronger magnifications produce less rotation, so that the information—magnification over either eye axis 180° and the rotation of the target plane in degrees plotted graphically—becomes an S-curve. From the center of the S-curve and the slope of the line determined by target rotation due to 2 percent, axis 90° over either eye, aniseikonia is determined.[6]

The Oblique Aniseikonia Experiment

An analogous experiment was performed on subject P. W. M. with oblique meridional size lenses and is summarized in Figure 5. Tests were made twice daily on the space eikonometer. Only the "delta," the amount of oblique aniseikonia, is recorded here. Axis 90° and axis 180° remained; 0.0 percent plus or minus 0.4 percent, and O.S., 0.8 percent plus or minus 0.7 percent respectively. Oblique aniseikonia is of clin-

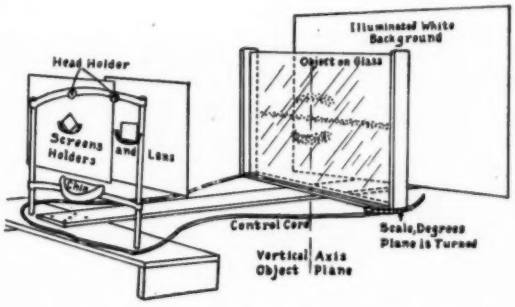

Fig. 4 (Miles). Schematic drawing of the frontal-plane apparatus used for measuring aniseikonia. Size lenses are placed at axis 180° before one eye magnifying from 1.0 to 14 percent, and the object plane is adjusted to appear perpendicular to the median plane of the head.

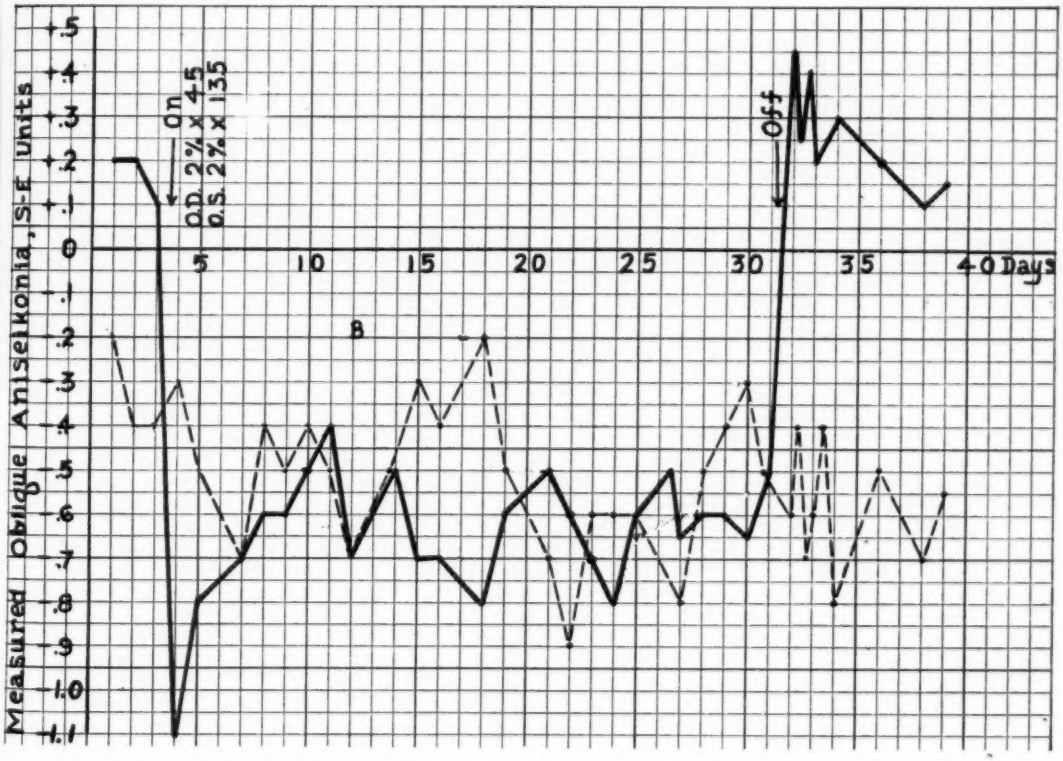

Fig. 5 (Miles). The solid line indicates the amount of oblique aniseikonia in subject P. W. M. when wearing oblique meridional size lenses as measured on the space eikonometer. The broken lines indicate simultaneous cyclophoria in the same units, apparently unaffected by the glasses. (B) indicates when partial subjective adaptation occurred.

ical importance in patents with oblique astigmatism.

In this experiment, the oblique lenses caused much more discomfort than did the axis 180°. The prescription was O.D., 2 percent, axis 45°; and O.S., 2 percent, axis 135°. This is equivalent in its effect on cyclophoria and spacial distortion to a dioptric strength of 2.5D over each eye at the same axes, placed at an eyewire distance of 9 mm. It is well known that patients wearing glasses for such astigmatism are seldom comfortable. The glasses worn in this experiment were afocal, and did not blur the vision.

In Figure 5, the solid line is the oblique aniseikonia as measured on the space eikonometer with actual strings at 10 feet. The broken line is cyclophoria measured in the same units under the same target conditions by means of a half-silvered mirror and additional test targets thereby projected into the scene.[7] It is seen that cyclophoria at the beginning was recorded at −0.2 and −0.4 (degrees total excyclophoria) but increased to about −0.6 percent plus or minus 0.3 percent during the experiment at the same time that adaptation to oblique aniseikonia as expressed by the solid line occurred. The persistence of cyclophoria after removal of the glasses is not understood.

Subjective symptoms

Such glasses, like plus cylinders with axes converging above, put strain on the superior oblique muscles. Each of the 28 days the glasses were worn, there was eye strain with a constant almost overwhelming desire to remove them. The pull on the superior obliques would have been decreased by adjusting the axes nearer either the vertical or horizontal. Adjusting the axes to diverge above would shift the pull to the inferior obliques which are normally much stronger and more efficient. The eyestrain from the experiment was never sufficient to prevent daily work, although concentration and study were difficult.

There was distortion, extreme at first, which was not much improved after a week. Even after 28 days, there was little subjective adaptation. The distortion, like that with axis-180° magnification, was limited to objects within about 40 feet. Geometrically, the distortion (for instance the rotation of a frontal plane in degrees) should increase approximately with the square of the distance.[8] Practically, this increase is not important because beyond about 40 feet monocular clues to depth perception begin to predominate. The question has been raised in the use of the space eikonometer whether the front vertical strings can be made equidistant to the observer without making the rear vertical strings unequal, and vice versa. In practice with the target at 10 feet, the difference is imperceptible. Due to the relatively small interpupillary distance, steropsis itself is limited to from 100 to 600 yards.[9]

At first, buildings and walls looked too tall, and seemed to lean toward me. Persons nearby seemed tall and overbearing with long spindly legs. On looking down at a grassy lawn, I seemed to be standing in a depressed area or trough extending laterally. On standing in a field of barley, this trough was deeper, and on changing the visual direction, the trough rotated causing everything to wave like the surface of the ocean. Walks through the woods were very unpleasant due to a feeling of unreality and even fear. The brush seemed to close in about the head. Persons with claustrophobia should be tested for this type of aniseikonia!

At badminton, I missed the shuttlecock by a foot or more at first. After some practice, I did fairly well.

I did not try to drive the car with these glasses on, but it has been done. A. Ames, Jr., reports,[8] "In driving an automobile . . . there is only the very slightest or no apparent tipping of the road. However, with the glasses one feels elevated, almost as if sailing along 10 or 15 feet above the road." With the meridional size lenses each rotated

90 degrees, "The glasses have the opposite effect, as if one were driving in a child's cart with his feet almost dragging on the road. Contrary to what might be expected, movement of the car enhances these effects."

Additional remarks

These subjective findings are rarely described by patients suffering from aniseikonia until glasses correcting the aniseikonia are worn. Patients then complain of distortion for a day or so. Clinical experience shows that when the full amount of aniseikonia measured on either the ophthalmo-eikonometer or space eikonometer is prescribed, the correction measured again at any later date remains consistent. One need not overcorrect or undercorrect aniseikonia.

Prof. R. E. Bannon told me of an instance "in the old days" when every patient so unfortunate as to have measurable aniseikonia was forthwith corrected by glasses. By some fluke, the size lens was placed over the wrong eye. The patient was I. C. F., Ph.D., who wore about 1.5-percent magnification on the wrong eye, thereby increasing what error she had by 1.5 percent. On reëxamination three years later, she was still wearing the glasses daily, and still showed the 1.5-percent error measured originally.

However, not all healthy individuals can wear experimental size lenses. A young physicist wore my O.S., 3-percent, axis-180° glasses for one-half hour, and went home with a splitting headache that lasted several hours.

Summary and Conclusions

This paper describes experiments in which meridional magnifying lenses were worn for prolonged periods of time. Subject P. W. M. wore 3-percent, axis-180° lens over the left eye for 25 days, changing immediately to 4-percent, axis-180° lens over the right eye for 37 days. Later, he wore O.D., 2-percent, axis 45°, and O.S., 2-percent, axis 135°, for 28 days. Subject E. J. B. wore 2-percent, axis 180°, O.D., for 23 days. With the axis-180° glasses, there was slight discomfort, occasional mild headaches, and marked distortion of objects in space. The distortion disappeared in about five days, except in surroundings devoid of perspective or rectilinear detail.

With the oblique aniseikonic glasses, there there was constant eyestrain and distortion of objects in space which was reduced in ordinary surroundings, but never did completely disappear. Excyclophoria produced during the experiment was measured and was found to increase an amount approximately equal to the amount which measured oblique aniseikonia decreased during the first two days. In other words, the compensation process which decreased subjective distortion involved increase in the cyclophoria.

The adaptation process otherwise is thought to involve a suppression of the objectionable parts of the image of the recessive eye. The parts suppressed are those slightly disparate due to the interpupillary distance. Since this disparity is necessary to stereopsis, this function becomes defective in aniseikonia, and monocular clues are given greater weight.

There is no real adaptation to aniseikonia. When an individual is forced to rely on stereopsis as in the space eikonometer, or when aniseikonia is measured by comparing actual visual angles as in the ophthalmo-eikonometer, aniseikonia is found to remain the same indefinitely.

There is apparently a time factor involved in measuring aniseikonia, which may explain some of the variability and "adaptation" shown in these experiments. In order to measure the total amount of aniseikonia present on either the space eikonometer or the ophthalmo-eikonometer, determinations must be repeated until, finally, memory factors, and habitual favoring of monocular clues over stereoscopic have been overcome.

Use of the space eikonometer is good training to develop stereopsis, since monocular clues are almost entirely eliminated.

In these experiments, the space eikonometer measured a larger portion of the artificial aniseikonia known to be present than did the ophthalmo-eikonometer. In one instance, the use of homatropine cycloplegia made the ophthalmo-eikonometer more accurate. Neither of these findings apply to clinical tests where more time is taken.

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Historical Miniature

The word "phoria" is associated with a multitude of concepts of binocular coördination. It was introduced by von Graefe to designate potential strabismus. He had observed that, with many pairs of eyes which were obviously free from strabismus, the double images induced by a vertical prism were also displaced laterally.

SIZE OF LINE IN THE MADDOX-ROD TEST*

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This study is one of several (Scobee and Green[1]) concerned with the effect of variables in testing technique on the measurement of heterophoria with the Maddox-rod test. The particular variable studied in this report was that of the size of the line of light as seen through the Maddox rod in measuring heterophoria at a testing distance of 13 inches. It is a well-known fact that different examiners using the same test of heterophoria on the same individual may get different results. In a search for possible explanations of this variation, several possible variables have already been studied. These include: examiner variation, test used and its correlation with the cover test, amount of illumination in the testing room, color of the Maddox rod used, the eye before which the Maddox rod is placed—that is, dominant or nondominant—in performing the test, and individual subject variation from day to day.

METHODS FOR MEASURING

TEST LIGHTS USED

It was believed that since different examiners may use different sized muscle lights at the 13-inch testing distance and produce varying sized lines as seen through the Maddox rod, any possible effect of this size difference should be determined. In the beginning, three test lights were used. One was an ophthalmoscope with a May-type head removed; a second was a flashlight fitted with a solid diaphragm in which a hole 1.0 mm. in diameter had been bored; a third was a Welch-Allen ophthalmoscope with the head removed. The May-type ophthalmoscope gave a broad line image when viewed through a white Maddox rod. The 1.0 mm. light gave a narrow, sharply delimited line. The Welch-Allen ophthalmoscope light had such a comparatively large filament and resulted in so many lines of varying brightness when viewed through the Maddox rod that it had to be discarded because it was too confusing to the subjects.

TESTING DISTANCE

All measurements were made at a testing distance of 13 inches with the testing technique previously described (Scobee and Green[1]). In brief, both lateral and vertical heterophoria were measured at 13 inches with a white Maddox rod placed before the right eye of all subjects. For lateral hetrophoria, a Risley rotary prism mounted on a phorometer (American Optical Company) was placed before the same eye as the Maddox rod; the rotary prism had a total strength of 30 prism diopters and was calibrated in units of 1 prism diopter. For vertical heterophoria, the Maddox rod was used in conjunction with a Stevens phorometer mounted on the same instrument; the prisms were split before the two eyes and had a total strength of 2 prism diopters, being calibrated in 0.2 diopter units. Thirty-two subjects were tested.

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Results of measurements

Lateral heterophoria

The average heterophoria was −4.89 prism diopters (exophoria) when the source of the light was large (ophthalmoscope) and −4.87 prism diopters when the source of light was small (flashlight). The difference of 0.02 prism diopters is not statistically significant, the standard error of the difference being 0.398 prism diopters ($t = 0.05$, $df = 31$, $P > 0.90$).

Two tests were made of the justification for using the statistical method of comparing a mean difference with its standard error. The first of these was to see if the frequency distributions of heterophoria under the two conditions of large and small sources of light were approximately normal in form. The distributions were shown to be sufficiently symmetrical and bell-shaped to justify the assumption of normality. The second test was to see if the variances of the two distributions were the same within sampling limits. These variances were found to be 25.67 square prism diopters for the large light source and 25.72 square prism diopters for the small light source. The ratio of these two variances, $F = 1.002$, indicates they are not significantly different as judged by the method of Morgan[2] for comparing variances of correlated series.

The heterophoria determinations under the two conditions of large and small light sources are highly correlated, $r = +0.90$. This is sufficiently high to indicate that knowledge of a heterophoria reading made under one condition will be equivalent for most practical circumstances to a heterophoria reading made under the other condition. Of the 32 subjects examined, only one had as much as 6 diopters difference, a shift from −14 prism diopters with the large light to −8 prism diopters with the small light. Two subjects had shifts of 4 prism diopters, one from +4 to +8, the other from −6 to −10. The remaining 29 subjects gave readings with differences of 3 prism diopters or less.

Vertical heterophoria

The average heterophoria was −0.03 prism diopters with a large light source and +0.03 prism diopters with a small light source.

The difference of 0.06 prism diopters is not significant when compared with its standard error of 0.12 prism diopters, ($t = 0.48$, $df = 31$, $P = 0.70 - 0.60$). The correlation coefficient was +0.85 and this is high enough to permit substitution of the large light for the small light reading or vice versa.

The same two tests for the justification of using the above test of significance were made. The distributions were approximately normal in form and the variances, 0.664 and 0.660, were homogeneous.

TABLE 1

Average heterophorias for 32 subjects for two sources of light*

Source of Light	Lateral Heterophoria (in prism diopters)	Vertical Heterophoria (in prism diopters)
Ophthalmoscope (large)	−4.89	−0.03
Flashlight (small)	−4.87	+0.03
Difference	0.02	0.06
Standard error of difference	±0.39	±0.12
Coefficient of correlation	±0.90	±0.85

* Note: − =exophoria; + =esophoria.
− =left hyperphoria; + =right hyperphoria.

Conclusions

There is no significant difference in the measurement of lateral and vertical heterophoria with the white Maddox rod at a testing distance of 13 inches when the size of the line as seen through the Maddox rod is either large or small. This means that any variation in heterophoria measurements in the same individual by different examiners using the Maddox-rod test at 13 inches cannot be considered due to any variation in the size of the line produced by varying sized muscle lights. Such a conclusion seems justified as long as the size of the muscle light used is within the limits of those included in this study.

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REGENERATION OF THE CORNEAL STROMAL CELLS*

I. Technique for destruction of corneal corpuscles by application of solidified (frozen) carbon dioxide

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Regeneration of the cornea has been extensively studied in the past. The origin and mode of replacement of the epithelium, endothelium, and Descemet's membrane is clearly understood.[1,2] There is still, however, a difference of opinion about the origin of the corneal stromal cells. A detailed review of the literature on this subject will be published at a later date. The purpose of this paper is to report the technique of a method for studying the regeneration of stromal cells which appears to be superior to the methods previously used by other investigators in this field.

Previous experimental methods

The essence of the previous methods is as follows. Salzer[3] made incisions into the

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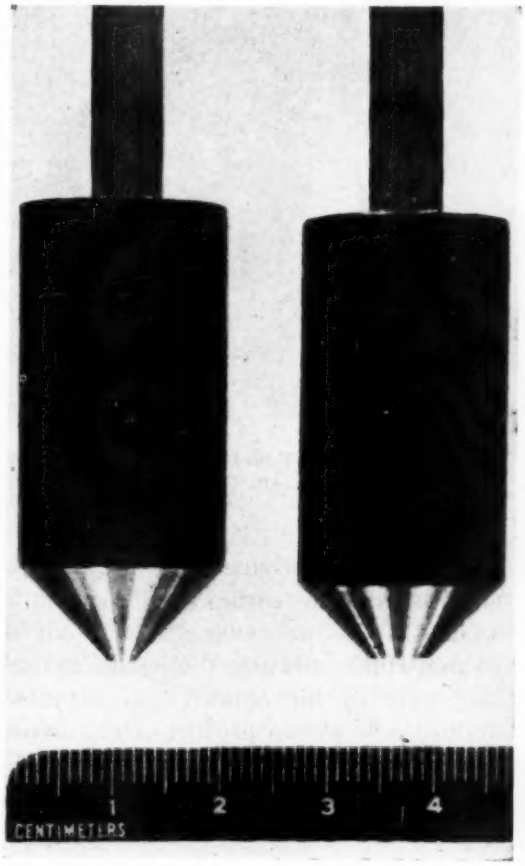

Fig. 1 (Maumenee and Kornblueth). Solid brass applicators.

cornea with a trephine and concluded that the new stromal cells were derived from the epithelial cells. Hanke[4] made perforating and nonperforating incisions into the cornea with a trephine. Wolfrum and Boehmig[5] used a knife to make nonperforating incisions into the stroma from the posterior surface of the cornea and perforating wounds from the external surface of the cornea. Hanke and also Wolfrum and Boehmig were of the opinion that the newly formed cells were a product of the division of the uninjured corneal corpuscles. Pullinger and Mann[6] destroyed the stromal cells in the center of the cornea by applying a small drop of liquid dichlorodiethylsulphide (mustard gas) to the cornea. The authors could not obtain definite proof, but they thought that the new stromal cells probably were derived from wandering macrophages. While these methods of attack are useful in the studies of the regeneration of the corneal stromal cells, they entail certain disadvantages in studying the regeneration of the corneal corpuscles in clear corneas. External and internal incision into the stroma disrupt the corneal lamellae, allow the ingrowth of epithelium or endothelium, open a portal for the entrance of secondary infection, and destroy only a few stromal

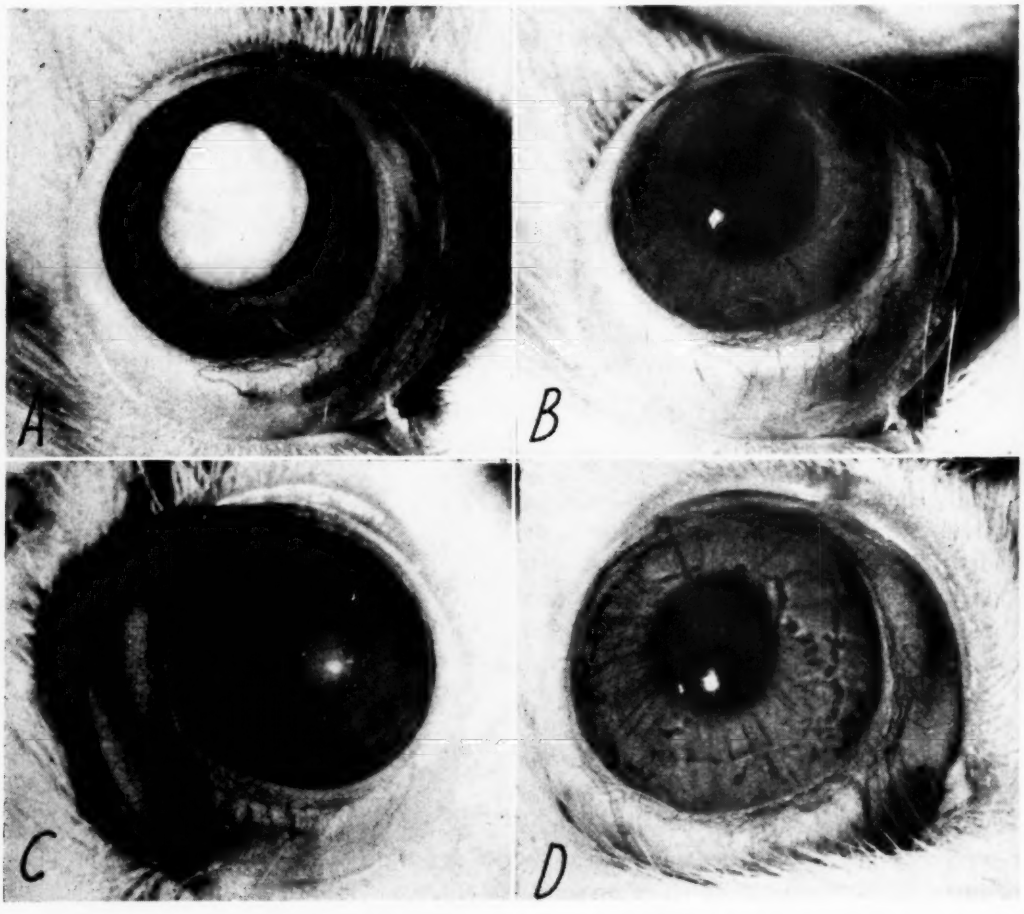

Fig. 2 (Maumenee and Kornblueth). (A) Rabbit cornea immediately after application of brass rod (—78°C.) for 5 seconds. (B) Same cornea 30 seconds later. (C) Same cornea 12 hours later. (D) Same cornea 7 days later.

cells. Lesions produced by liquid "mustard" eliminate these complications, but they also have some disadvantages. Liquid "mustard" is hard to obtain in some laboratories, it produces considerable inflammatory reaction, and the dosage is difficult to control.

Method used in this study

The method here reported consists in destroying the corneal stromal cells without disrupting the corneal lamellae or producing a severe inflammatory reaction.

Materials. The materials used are solidified (frozen) carbon dioxide (dry ice), absolute alcohol, and a solid metal rod. The solid metal rods are used because they are good temperature conductors, they will maintain the desired degree of coldness for a sufficient period of time to allow completion of the experiment, and they will produce a standard area of freezing. In our experiments solid brass rods, 19 mm. in diameter and 45 mm. long, were used. The end of the rod which was applied to the cornea was shaved down so that the area of contact was a circle 2 mm. in diameter on one applicator, and 6 mm. on the other (fig. 1).

The applicators are cooled to −78°C. by immersing them for a short while in a beaker of absolute alcohol containing "dry ice." The alcohol is removed from the applicators with a dry towel before placing them in contact with the cornea.

The animals are given a general anesthesia and the eyes are proptosed from the socket or fixed with forceps so that an accurate application of the rod to the cornea can be made. The lesions may be produced in the center or the periphery of the cornea. The area of the lesions can be varied from a circle, 2 mm. in diameter, to the entire cornea.

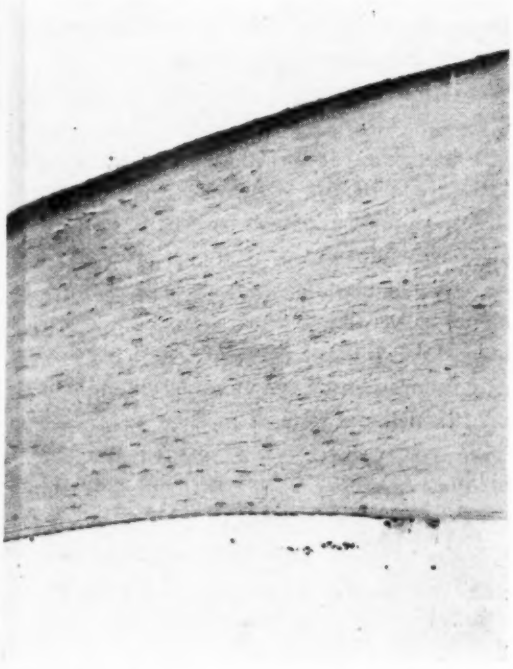

Fig. 3 (Maumenee and Kornblueth). Rabbit cornea 12 hours after application of 6-mm. brass rod (−78°C.) for 5 seconds. This view shows the margin of the lesion. (Histologic section. Hematoxylin-eosin stain. Magnification ×200.)

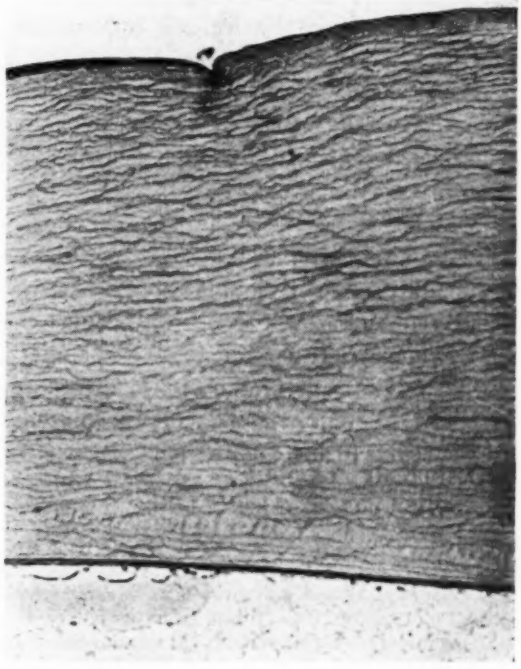

Fig. 4 (Maumenee and Kornblueth). This shows a slightly more central area of the same lesion as in Figure 3.

Application of the brass rod to a rabbit's cornea for 3 to 5 seconds freezes the full thickness of the cornea for 15 to 25 seconds (fig. 2A).

Appearance of cornea. After the cornea thaws it appears clear, although a very slight haze is present (fig. 2B). Two to three hours later the cornea becomes hazy due to edema of the stroma (fig. 2C). This edema persists for a varying length of time depending on the duration and area of the freezing. When the edema subsides, the cornea resumes its normal clarity and appearance on gross and slitlamp examination (fig. 2D). Blood vessels do not invade the cornea following central or peripheral lesions but they do invade the stroma following freezing of the entire cornea. Secondary infection does not occur except in some lesions of the entire cornea where there is persistent edema and eventual sloughing of areas of the stroma.

Histologic study. On histologic examination, all of the cells of the cornea including the epithelial, stromal, and endothelial cells are destroyed in the area that has been frozen. The corneal lamellae, however, do not appear to be damaged by this procedure. Twelve hours after freezing the cornea is edematous, and all remnants of the stromal cells have disappeared in the area of the lesion (figs. 3 and 4). It is remarkable that so few polymorphonuclear cells invade the cornea in spite of the extensive destruction of the stromal cells. After 72 hours most of the polymorphonuclear cells have disappeared from the cornea, and stromal cells show definite replacement in the periphery of the lesion. During the next few days, the stroma assumes an entirely normal appearance on histologic examination.

Summary

It has been found that freezing various sized areas of the cornea to −78°C. for 3 to 5 seconds produces a very convenient lesion for the study of the regeneration of the corneal stromal cells. These lesions do not disrupt the corneal lamellae, are not prone to secondary infection, and cause very little inflammatory reaction. After the lesions have healed the cornea is clear.

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TREATMENT OF PHORIAS IN ADULTS*

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Treatment of phorias in adults is highly important, extremely gratifying in the results obtained, and, due to the wide variety of problems presented, is the most interesting procedure in orthoptic work.

SYMPTOMS OF PHORIAS

The importance of treatment cannot be too highly stressed since the patients in this group suffer the greatest amount of discomfort of any persons having an "ocular muscle imbalance." Frequently the patient so afflicted is the one who goes from "doctor to doctor" with the hope of getting a pair of glasses that will be just right and will relieve the strain, fatigue, or particular discomfort to which he is subject. A thorough case history should be taken and the patient encouraged to talk about his eyes. This will not only make possible a better diagnosis but give the patient confidence and the feeling of being understood which psychologically will aid in relieving his symptoms.

Burning, itching, difficulty in focusing or in altering the focus quickly may be some of the symptoms of heterophoria; while severe headache, especially after close work, even the "one-sided" headache with nausea typical of migraine, may be experienced. Other symptoms can be blurring of vision approximating diplopia or actual diplopia. Some patients complain of nothing but inability to read longer than five minutes.

PROBLEMS IN TREATMENT

The first step in treating phorias is to do a careful refraction under cycloplegic, to be followed at a later visit by a Maddox-rod test on a phorometer and, if possible, a major amblyoscope.

Livingston suggests that heterophoria should be divided into two primary groups: (a) inherent, and (b) acquired; and that it is necessary to speak of that which is (a) symptom free, and (b) symptom producing.

He also states that ocular imbalances and their sequelae follow no happy and consistent pathway. Certainly this is true. A cover test for near may show a deviation of approximately 26-degrees exophoria, yet it will measure only 12 degrees on a Maddox-rod scale, and a patient whose measurements are orthophoric may be found to have several degrees of deviation when the cover test is applied. Measurements may vary in morning and late-afternoon readings and on subsequent days, while three consecutive tests may reveal three widely variant responses. These results, rather than being confusing, should lead to a more comprehensive understanding of this patient's problems, as will be shown later.

After the muscle-balance and fusional-reserve tests, including the near point of convergence have been taken, a decision must be made as to the patient's need for treatment. It is a very simple decision. Phorias are important not because of their degree, but because of the symptoms which may arise from this condition. Therefore, the patient who is "symptom free," regardless of how large the degree of heterophoria, is best left alone. To attempt treatment with the hope of achieving orthophoria, or of preventing future symptoms might well prove disastrous both physically and psychologically by giving him an "awareness" of his condition. However, these same tests

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should prove determining factors in the prescribing of glasses.

Prescription of glasses

A hypermetrope with esophoria may, or may not, be fully corrected; whereas, one with exophoria should be undercorrected. A myope with exophoria is more comfortable, as a rule, with full correction although occasionally one is found with a high degree of myopia who will not tolerate this, thus necessitating the sacrifice of vision for comfort. A myopic patient with esophoria will almost invariably experience discomfort for near work which may usually be relieved by the use of bifocals or reading glasses although he is not presbyopic. To disregard the importance of phoria measurements when prescribing glasses may cause one who is orthophoric to become esophoric or exophoric, as the case may be, and may explain the complaint of the patient who later returns saying that although he can "see just fine with the new glasses he cannot stand to wear them." Therefore, it cannot be too strongly emphasized that a careful refraction is the first step in the treatment of heterophoria. Indeed, in some cases it is the only treatment that is required.

Importance of general health

Next in importance is attention to the general health. There is no doubt that the physical condition of a patient may be the dividing line between "symptom free" and "symptom producing" heterophoria. One whose condition has previously not caused symptoms may do so following an illness or peroid of over fatigue. Lack of sleep, toxicity, foci of infection, and especially anemia are all debilitating factors and should have careful attention. Not to be overlooked as a cause for eye discomfort is the habitual use of intoxicants. The prognosis in such cases is good with appropriate treatment, and so far as possible, the elimination of the various causes.

An occupation which requires constant use of the eyes for near work may cause symptoms to a person who previously has been most comfortable. Commercial artists, draftsmen, printers, and bookkeepers are in high number on this list. With "fusional reserve" built up, however, and with correction of the "convergence insufficiency" that is usually acquired in such cases, it is rare for these patients not to be made perfectly comfortable again, and equally rare for it to be necessary for them to change their type of work.

Psychologic problems

The last, and certainly the most difficult problem, is the psychologic condition encountered in some individuals. These patients are the ones referred to earlier in this paper. Their measurements may be small but their symptoms are excessive. The inconsistencies, which are so typical of these patients, must not be confused with those of patients whose readings on the Maddox rod are far less than a cover test would indicate—discrepancies that may be explained by the inherent desire for binocular vision and a large "fusional reserve."

Far from being placid individuals, the patients who present psychologic problems are usually introspective, nervous, and of the anxious type. A frequent factor is unhappiness, either in the home or at work, but whatever the origin of the disturbance, the prognosis is not good unless the underlying cause is removed or at least recognized. Once this is done, these patients usually respond amazingly well to treatment.

Although it does not belong to the heterophorias, there is another condition so closely related in many aspects as to deserve attention. The symptoms are rather vague although the patient is most insistent about not being comfortable either with glasses or without them.

Examination will usually disclose a relatively small refractive error and near orthophoria. Upon closer questioning these patients reveal that they have consulted sev-

eral doctors about their nerves only to be told that they are in good condition. When further pressed for information regarding their eyes, they may state that objects or people will look much closer to them at one time than another, or perhaps smaller and then larger. If taken to a window where they may view a nearby brick wall they may also state that the bricks are not consistent in size and shape. Certainly an aniseikonia test is indicated here and will usually reveal an overall or meridional image size difference of one half to three percent. Correction by an iso-iconic lens is usually successful in "curing" this patient's symptoms. Fusional exercises are also given as aids in stabilizing fusion and increasing the accuracy of stereoscopic vsion.

Aim of treatment

The phorias fall into four groups: esophoria, exophoria, hyperphoria, and cyclophoria. Before considering the treatment for each of these conditions, however, it is well to understand the aim of such treatment.

The aim of treatment is to relieve the patient's symptoms, not to achieve orthophoria. Although orthophoria is achieved in some cases, in other cases the measurements may show little or no improvement.

Maddox stated with regard to the treatment of heterophoria: "It is impossible to make rigid rules, since it is not a disease, but only a symptom to collate with other symptoms, as well as the patient's temperament, and the constitutional blood and nerve state." This being true, I can think of no instance where imagination and a real desire to help may be better displayed, for it must be remembered that one is treating individuals, not types.

Gain patient's confidence

The approach made during the first treatment is all-important. It can mean the difference between success and failure. Devoting a half hour to explaining the patient's particular condition, making him realize that an effort of will is absolutely necessary, assuring him of your understanding and sympathy will require tact and patience but will add much to the prognosis. Prepare the patient for discomfort, including nausea (especially exo's and convergence insufficiencies). After the confidence and coöperation of the patient have been won, follow up by showing enthusiasm when the first positive effects are demonstrated. Not infrequently this may be on the second or third visit.

Exercises for home work

All patients should be given motility exercises for home work—the six cardinal positions plus convergence, with emphasis on their weakest position. A 3-mm. white-headed pin inserted in the top of a pencil is ideal for fixation. Three 5-minute exercise periods a day are more beneficial than one 15-minute period. A fusion-stimulating exercise should also be given. Use a piece of red Kodaloid and a small light, preferably a pencil flashlight, and work from reading distance to infinity. (With the red Kodaloid held over one eye, the light must be seen "single" and pinkish in color). This exercise also aids in breaking up the suppression that occurs in a large percentage of heterophorias and is quite possible in all phorias on certain occasions.

Exophoria treatment

General treatment for exophoria consists of (a) correction of suppression, (b) good N.P.C., (c) prism convergence, and (d) good accommodation.

With a 3-mm. pin in a pencil held at reading distance, hand prisms may be placed (base out) before the eyes until an average of 15$^{\Delta}$ is reached. Each time the prisms are increased (by units of 2$^{\Delta}$) the patient should look alternately right and left, coming back to the pin each time, making sure it is "single and clear." This should be followed by fixing on the muscle light (at 20 feet) and, if possible, a light in the upper right-hand and upper left-hand positions

(about 7 feet high), and the same amount of prism convergence repeated as for near. An addition of 10$^{\Delta}$ each week is sufficient, with the maximum of 75$^{\Delta}$ reached usually by the seventh week.

Next the patient is placed at a major amblyoscope and careful attention given to suppression—both on convergence and divergence. Use of fusion slides with small central controls are best. Flashing is very helpful, after which convergence may be started. Side movements are important both from the standpoint of suppression and muscle coördination. The Stereo-Orthoptor is particularly well fitted to phoria treatment because of its automatic features. A Worth hand amblyoscope is also very good for homework at this stage because of its hand-eye coördination value.

Hand-eye coördination

Hand-eye coördination is of inestimable value and should be utilized in every possible way. These patients are usually slow to accommodate and change focus, therefore, at about the fourth visit two ink dots should be placed on a pin for home exercise. This requires accommodation as well as convergence (to be seen clearly) and looking from pin to small objects such as door knobs or light switches at distances of 5, 10, 15, and 20 feet builds up accommodation and the ability to change focus quickly.

Also, at about this time, physiologic diplopia should be taught and practiced. Later, it should be followed by bar reading. At the 7th or 8th visit a Keystone stereoscope and base-out cards may be borrowed for home training. The Ortho-fuser is also very good for home training. Usually 12 weekly treatments are required to complete this routine. The patient should have experienced some relief of symptoms early in the training but if treatments are stopped before completion, the symptoms are quite apt to return in a few months. Patients with exophoria are quite often poor readers, have a tendency to transpose, and are sensitive to light. This last, no doubt, explains why many have had tinted lens prescribed.

Standard of cure

At the conclusion of treatment the patient should be reëxamined. Usually the axis will have changed position a few degrees, with a decrease in the sphere—particularly plus but sometimes minus. In many cases of a small refractive error, glasses may be discarded entirely, and frequently presbyopic patients will require less addition. This I do not believe due to an actual change of refractive error (as tested by cycloplegic) but to the better seeing habits and skill developed, with most efficient use of extrinsic and ciliary muscles.

A standard of cure could be: (1) No symptoms, (2) prism convergence (70$^{\Delta}$ to 75$^{\Delta}$), (3) prism divergence (7$^{\Delta}$ to 8$^{\Delta}$), (4) good N.P.C. (25 mm.), (5) accommodation (Prince rule).

Esophoria treatment

General treatment for esophoria consists of: (a) Correction of suppression, (b) prism divergence, (c) good accommodation.

The esophoric patient usually complains of blurring or diplopia in the distance, with headache and discomfort for reading or near work. Change of focus is slow, probably due to effort of straightening visual axes. A poor near point of convergence also exists in many cases.

The first step in treating this patient is to teach him the difference between "accommodation" and "relaxation of accommodation." This may be done simply and quickly by placing a red glass, or red Kodaloid, before one eye while the patient fixes on the muscle lights (20 feet). Told to "clear" the lights he will find that they go much farther apart (homonymous). When told to relax and "blur" the eyes, the images will be much closer—sometimes even fusing.

After recognizing this fact he is told to see "clearly" while effort is maintained to

fuse the lights. A prism (base out) of the necessary amount to get fusion is placed before one eye. This is decreased, 1$^{\Delta}$ at a time, until none is required. Then starting with a prism, 1$^{\Delta}$, base in, increase to an approximate 7$^{\Delta}$, base in. An aid to effort will be gained by having the patient look "up and over," pulling hard, then back at the light when fusion becomes difficult or unstable.

On the major amblyoscope fusion slides are joined at the patient's angle—the patient again having been told to see "clearly." Special attention must be given to suppression and side movements after which divergence is started. Amplitude is usually found to be low, necessitating the teaching of good convergence as well. After a maximum of divergence has been reached—approximately 7$^{\Delta}$—the patient should be taught to disassociate accommodation from convergence. This may be done by inserting −1.0D. sph. and clearing the picture at "O" followed in turn by −2.0D. and −3.0D. spheres.

A Keystone stereoscope with base-in cards is excellent for home use. The Orthofuser or Remy separator may also be used. Physiologic diplopia and bar reading are invaluable aids—both to stabilization and accommodation—but must not be attempted too soon. Divergence is not a natural anatomic feature such as convergence; therefore, progress is much slower and relapses more frequent. The myopic patient presents an even greater problem but may still be considered a good orthoptic risk.

Standard of Cure

A standard of cure could be: (1) No symptoms, (2) prism convergence (50$^{\Delta}$), (3) prism divergence (5$^{\Delta}$ to 8$^{\Delta}$), (4) good N.P.C. (25 mm.), (5) good accommodation (Prince rule).

Pure Hyperphorias

Pure hyperphorias are rare. The patient complains at times of vertical diplopia, is usually nervous, and is more conscious of his cosmetic appearance than either the esophoric or exophoric patient.

General treatment is the same as for the preceding phorias. On the major amblyoscope the patient should join the fusion slides with the least possible vertical adjustment. Again special attention must be given to central suppression, after which supravergence and infravergence are stressed. Both supravergence and infravergence are much improved by good prism convergence and prism divergence. The Orthofuser or a Keystone stereoscope, with both base-in and base-out cards, may be given for home exercise; the use of red Kodaloid is particularly helpful in these cases, with physiologic diplopia and bar reading taught and practiced as early as possible. It is entirely possible to correct a hyperphoria of 14$^{\Delta}$ and make the patient comfortable. Many times a hyperphoria of small degree will be found in conjunction with an exophoria or esophoria, particularly the former, in which case, correction of the lateral will usually be all that is needed.

Cyclophoria

Cyclophoria is perhaps the least known and considered of the phorias. Symptoms are headaches, nausea, and possible head tilting.

In all cases of head tilt in which the patient is found to be orthophoric or nearly so, a careful test should be made to determine the possibility of cyclophoria. The Synoptophore is well equipped to detect and measure deviations as well as correct this condition. Fusion slides are joined at the necessary oblique, or torsion angle and the image gradually straightened while fusion is maintained. Hyperphoria may also be found in conjunction with cyclophoria; or, due to the effort to overcome hyperphoria, a resultant cyclophoria may occur. In either case the treatment is the same. Correct the hyperphoria.

Another cause of cyclophoria is oblique

astigmatism and, as Maddox has pointed out, "in uncorrected oblique astigmatism, corrective torsion becomes a life habit." When strong cylindrical lenses are prescribed, care must be stressed not only with regard to the correct axes but to the constant maintaining of proper adjustment.

Convergence insufficiency

Convergence insufficiency is not considered a member of the heterophoria group but deserves special attention as it probably causes more discomfort than any other one condition. It is also, as a rule, one of the easiest to correct. Convergence insufficiency may be acquired by long hours of close work, fatigue, or lack of muscle tone due to ill health. Frontal headaches, tired eyes, and at times an actual aversion to anything requiring near vision, are common symptoms. These patients will be found, in most instances, to be monocular readers. Indeed, many of them are aware of it, either when having it pointed out to them, or by volunteering the information. Certainly one whose near point of convergence is 40 cm. (16 inches) would be unable to maintain fusion.

Suppression —deep suppression—is the main characteristic of this condition and must be borne in mind throughout the entire treatment. It is even advisable to use a red glass, or red Kodaloid, over one eye when taking the N.P.C.

Complete motility exercises should be given in addition to convergence with a pin. Again the red Kodaloid should be used (during convergence) as it is entirely possible to get muscular coördination and continued suppression. The two dots placed on the pin are very good, as an alternate exercise, because these patients need incentive and this in turn helps accommodation. Vergence exercises on the Synoptophore or Stereo-Orthoptor are the same as for a phoria, with particular attention to suppression. Physiologic diplopia and bar reading should be taught as soon as good convergence permits, and last, but of equal importance, is the teaching of voluntary convergence. Usually 4 to 6 treatments are sufficient.

The incorporation of prisms in glasses is a method of treatment which we do not recommend. It has been our observation that an additional amount must be added from time to time and after a few months, or years, the patient is unable to tolerate the added weight and chromatic aberration of prisms strong enough to correct a phoria of any considerable degree. Before starting orthoptic treatment of patients wearing prisms, new glasses are prescribed, without any prism whatever.

A practice which should be given careful consideration is the one of patching where a phoria exists. There is grave danger of a resultant tropia. If you feel that it is absolutely necessary, then a week or 10 days should be the limit of time, with frequent observation.

It has been a very great pleasure recently, either to reëxamine or to talk with patients who were given orthoptic treatments during the past six years. In none have the symptoms returned, except fleetingly when tired, and not one, so far, has required a refresher course.

Conclusions

1. Phorias are important.
2. Phorias respond readily to treatment.
3. The aim of treatment is not orthophoria, but the relief of symptoms.
4. This group is composed of your most satisfied patients.

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TISSUE RESEARCH AND CELL CULTURE OF THE CORNEA

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Although we can be proud of the progress that has already been made in keratoplasty (especially in the use of donor material), our final aim is to create conditions whereby the host cornea will accept the transplanted material completely so that a later cloudiness, which so often develops in transplantations of the same species but of different blood groups, will not mar the end result.

In view of the fact that the literature on keratoplasty reports unsatisfactory results in a majority of cases, it would seem pertinent to question why the implant (of the same species) becomes cloudy and to ask whether or not colloidal chemistry might offer an approach to the problem. It would seem that a study of the chemical and physical (such as swelling) factors of the albumin which form the base of horny tissues might suggest a solution to some of the difficulties of corneal transplantation.

It may be that the donor cornea, especially the cadaver cornea, loses its own specific characteristics under the effect of artificial culture and becomes an indifferent piece of tissue that cannot be changed by later assimilation with the host tissue. As far as I can learn from a study of the literature, the following possibility has never been fully considered.

If the transplant is kept alive by means of slow-growing cell culture, and if the plasma content of the blood of the recipient is increased before the transplant is made, it might be that the original structure of the albumin of the transplant could be so changed that it would gradually become assimilated with the albumin of the host.

Loehlein was of the opinion that if corneal tissue were carefully kept alive by means of culture passages, conditions might be created which would permit a gradual and delicate balance between the host and the donor tissues. Loehlein and Salzer both came to the conclusion that not only did corneal tissue (carefully kept alive) of dead human bodies (in my opinion the best means to accomplish this is through sterile culture) make the best transplants and cause the least reaction with the surrounding tissues, but also that homologous tissue furnishes the most favorable conditions by providing a frame for the gradual regeneration of tissue.

According to Widal, corneal cloudiness results chiefly from "protein shock" or the coming together of two antagonistic colloids. We know that the albumin of the plasma reacts to the sudden introduction of chemicals or to plasma of a foreign type. In his discussion of the so-called "crises of blood types," which led to his observations on the process of protein shock, Widal says: "The shock is not confined to the colloids of the blood; it also exists in all the plasma, making itself felt even in the plasma colloids which constitute our cellular elements."

Widal's highly interesting conceptions of the colloidal processes are not only applicable to the colloids of the blood, but they apply wherever proteins are involved. In my opinion, these observations can provide an explanation of the cloudiness of corneal transplants.

It is now pertinent to mention the changes which it is possible to produce in the chemical structure of albumin and which may account for the assimilation of the donor albumin with that of the host organism.

We learn from the earlier publications of Obermeyer and Pick that the specific grouping of the same kind of albumin molecules is influenced by the various arrangements of albumin atoms. These authors, by introducing nitroiodine and diazo groups, succeeded in changing the so-called "original" structure of the albumin into a "constitutional" one. The "host" organism of the injected animal reacted only upon the changed

albumin in a "specific" manner and while the species characteristic was completely extinct.

Since these structural changes in the albumin molecule depend upon the regrouping of the albumin atoms in the chemical ring, it would be the aim in cell culture for corneal transplantation to produce, by influencing the chemical structure of the molecule, such gradual changes as would be equivalent to an assimilation of both kinds of albumin. Evidence as to whether, as a result of cell culture, this assimilation was taking place would be cloudiness appearing more or less rapidly, for, after all, growth in cell culture means a fundamental transformation.

It would seem that further assimilation could be induced by changing certain physical factors affecting albumin, such as, for example, swelling. So far, the nature of this factor of swelling is not completely understood, but its uniformity in the healthy cornea presupposes the existence of a most accurate and complete method of regulation. The forces which bind together water and the colloidal albumin molecule are extremely great. They represent the so-called swelling pressure or, according to Schade, the colloidosmotic pressure.

In the literature on corneal transplantation, it has been remarked that, only after keratoplasty in interstitial keratitis, has perfect clearing been observed and then with deeply located, thick, cloudy specks. Imre is of the opinion that this is due to colloidochemical changes of the corneal tissue, changes which are reversible.

Thoenes, an investigator of tissue colloids, followed the laws of water binding that had been examined by Rübner, Müller-Thurgau, H. W. Fischer, and Bobertag. He believed that there are changes which have something to do with a progressive growth, and he says, "With progressive growth not only the total amount of the tissue water is diminished, but also a change in the physical structure of the tissue colloids takes place at the same time, leading to a diminution of the water bound to the protoplasm molecules."

These observations lead one to consider the possibility that, through the gradual growth of the cell culture in vitro, it may be possible to create such delicate methods of regulation as the eye afflicted with interstitial keratitis is capable of creating when a keratoplasty is performed upon it.

Another factor—that of the same blood group—which, in my opinion, may play a part in the clouding of corneal transplant has no importance according to the literature on the subject (Loehlein, Salzer, Imre, Nižetić, Magitot, and others).

Cell-culture experiments

The controversy as to whether the corneal graft continues to live as an independent tissue or is replaced by regenerated corneal tissue of the host has never been definitely settled by the ophthalmologists. Loehlein, Salzer, Bonnefon and Lacoste, Leonardi, and others believe that the transplant tissue never remains intact as such, but that it is replaced slowly and gradually by ingrowing tissue from the host. Filatov, Castroviejo, Leoz, Ortin, Ascher, Sommer, and others, however, are of the opinion that the graft, if remaining clear, will continue its existence as independent tissue.

In view of this difference in opinion, it seemed important to watch the growth of the different parts of the cornea. As a preliminary to my attempt to obtain corneal transplants that did not become cloudy, I examined the growth of the different layers of the cornea. Corneas of rabbits, guinea pigs, and chicken embryos were supplied by Dr. Vollmar of the Department of Tissue Research, Forschungs-institut für Chemotherapie, Frankfurt. Cadaver corneas were furnished by Professor Lauchle of the Pathological Institute.

We are indebted to Imre for some good reports on histologic findings in corneal transplants. His histologic specimens show again and again the typical corneal cloudiness. The epithelium is denuded, Bowman's

membrane is not recognizable, the upper layers of the cornea contain hyaline deposits—granular and filamentous calcium deposits—the substantia propria is replaced by coarse, granulated tissue streaked with vessels.

A specimen of clearly healed-in graft is rarely obtained. One specimen of a clear graft, mentioned by Imre, had been removed four weeks after transplantation because the inner layers had become opaque.

Imre says that the epithelial and border membranes were well preserved. The endothelium could be recognized behind Descemet's membrane. The epithelial layer consisted of 4 to 5 rows in those parts of the graft near the border. At the center, however, the epithelial layer consisted of only two rows.

In the cell cultures which I made, it was interesting to watch the growth of the epithelial layer. I was able to obtain particularly fine cultures from rabbit corneas. (Reports of typical cell-culture growth of the epithelium borders of rabbit corneas have been published by Dr. Vollmar.) I was best able to observe the growth of spindlelike fibroblasts in corneal cultures of chicken embryos.

Mention has been made in the literature for tissue research and cell culture of a stage in cell growth intermediate between epithelial cells and fibroblasts. It is supposed that these cells originate from the corneal endothelium. Since, however, I have never been able to find a detailed description nor illustrations of this cell growth, I shall describe my own observations in detail.

The best growth of this intermediate type of cells that I have observed was in cultures of human corneas removed immediately after death (fig. 1). I received these cultures in a sterile state; that is, after the whole conjunctival sac had been washed out with boric-acid solution. It would seem that the boric acid has some growth-inhibiting effects upon the culture, since growth is not so extensive after its use. Because it is necessary to obtain sterile cultures, it might

Fig. 1 (Hoof). Cell culture of a human cornea removed immediately after death. This shows 24-hour growth at 37° C. (Magnification ×130.)

be that better results could be obtained through the use of some chemicotherapeutic agent that would specifically stop the growth of the bacteria without affecting the tissues.

In experimenting with the culture of the corneas of guinea pigs upon plasma of a different species (rabbit), no growth whatsoever took place. When fresh embryonal extract and the plasma of chicken or fresh guinea-pig plasma and a sterile buffer solution were employed for infusion, a surprising growth of the cornea took place.

Microscopic examination of these cultures showed a multitude of shapes. Just as illustrations in a zoology textbook depict groups of Polymedusae, with the freshwater polypi stretching out their tentacles, or the footlike fringes of the Rhizopoda floating around a one-cell Algae, so the microscope revealed the cells in the corneal cell culture moving out of the substantia propria, at first being attached to it like fixed species of Infusoria and later like

Fig. 2 (Hoof). Cell culture of guinea-pig cornea. This shows 48-hour growth at 37° C. (Magnification ×130.)

freely swimming shapes which were small, rounded-off lumps of protoplasm extending themselves.

The growth advanced and kept on moving until the starlike, branched-out cells, with the long and small cores, and the long threads of the cell protoplasm formed a broad circle around the substantia propria. The protoplasm also contained fine granules. Only in a 48-hour culture of guinea-pig corneas (fig. 2) have I seen this protoplasmic picture.

(In their histologic structure, the cells of guinea-pig cornea do not differ greatly from human corneal cells. These typical cells, located in the substantia propria, are represented by clumsy, irregular, and much perforated protoplasm layers, with either broad or long-draw-out, thin anastomoses [Schaffer].)

Comment

These preliminary experiments in tissue culture show the possibility of growth of the parenchyma cells out of the substantia propria. They would seem to substantiate the theory that the corneal graft lives as an independent tissue within the host's cornea, as advanced by Marchand, Fuchs, Filatov, Castroviejo, Leoz, Ortin, Ascher, Sommer, and others.

3 Rheinblick Street.

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COMPARISON OF EYE CONDITIONS AMONG 1,000 READING FAILURES, 500 OPHTHALMIC PATIENTS, AND 150 UNSELECTED CHILDREN*

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This is a comparative study of the eye conditions of 1,000 poor readers, 500 ophthalmic cases, and 150 unselected children. The median ages of the groups were 9 years and 8 months, 11 years and 6 months, and 10 years and 8 months, respectively. The median intelligence quotients for the groups were 102, 103, and 109 in the same order. All of the cases were examined by the same doctor with the same equipment and the same tests. The routine was that of the usual eye examination plus tachistoscopic measurements.

Refractions were made without a cycloplegic in order to permit observation of the pupils' use of their eyes in the reading situation. Careful retinoscopic and subjective refraction was carried out in all cases. The tachistoscopic data were collected with an instrument and method described previously.[1, 2] There is a great deal of variation in the tachistoscopes now in use, particularly in such variables as test types, vocabulary level of test words, object size, reading distance, intensity of illumination, and so forth. Results obtained from different makes of tachistoscopes are not likely to be mathematically comparable and so *tendencies* rather than mathematical values are significant except when instrument and test conditions are identical.

The results of all tests were distributed in each group, and frequency and central tendency were determined for each. This was followed by comparison of the groups.

The poor readers and the unselected pupils exhibited closely comparable frequencies of vision below 20/30, while the frequencies of vision below 20/40 were fairly close but not so much so. Both groups differed widely from the ophthalmic cases, as might be expected. The frequency of amblyopia was somewhat similar in the reading failure and unselected groups but the incidence among the ophthalmic patients was about twice as great for the left eyes as in the other two groups.

The frequency of hypermetropia of one diopter or more was greatest in the ophthalmic group, 7 percent less in the reading-failure group, and approximately 30 percent less among the unselected. The incidence of myopia of one diopter or more was practically the same in the reading failure and unselected groups, while the ophthalmic patients presented twice as high a frequency. The unselected cases exhibited a somewhat higher frequency of hypermetropic astigmatism than the ophthalmic group, while the poor readers displayed a still higher incidence. The step up from group to group was only 2 percent, however, making the frequencies fairly comparable in all groups. The incidence of myopic astigmatism was the same throughout all classifications. Both the reading-failure and ophthalmic groups exhibited frequencies of anisometropia that were quite close and about twice that of the unselected group.

Exophoria of 6 prism diopters or more in distant vision was most frequent among the unselected and least among the reading failures, while exophoria of the same magnitude in near vision was greatest among the reading failures and least among the unselected. Esophoria of a similar amount was slightly more frequent in near vison than in distant vision among the reading failures and the ophthalmic groups, the former displaying lower frequencies at both distances than either of the others. Hyperphoria did not appear in the unselected group but occurred in distant vision only to the extent of 1 percent in the others.

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Convergent strabismus was more frequent among the poor readers than among the ophthalmic cases, while divergent strabismus occurred equally in these two groups. No oblique deviations were noted in either group. No cases of strabismus were encountered in the unselected group. The greatest frequency of fusion deficiency was found among the reading failures. It was less frequent among the unselected and least among the ophthalmic cases.

Right eyedness occurred most often in the ophthalmic group and 11 percent less frequently among the reading failures, with the incidence in the unselected group falling between the others. The incidence of left eyedness was closely similar in the reading-failure and unselected groups, with the ophthalmic group presenting a lower frequency. Ambieyedness occurred as frequently in the poor reading and ophthalmic groups; rather less so among the unselected children.

TABLE 1

FREQUENCY OF EYE CONDITIONS IN READING FAILURE, OPHTHALMIC, AND UNSELECTED GROUPS

Condition	Reading Failures		Ophthalmic		Unselected	
	Right	Left	Right	Left	Right	Left
Number of cases	1000		500		150	
	percent		percent		percent	
Vision						
Below 20/30	35	35	55	57	36	34
Below 20/40	19	19	38	40	13	14
Amblyopia	13	10	15	20	11	12
Refraction						
Hypermetropia of 1D. or more	43	43	50	50	13	12
Myopia of 1D. or more	4	4	8	8	4	3
Hypermetropia Astigmatism of 1D. or more	6	6	2	2	4	4
Myopic astigmatism of 1D. or more	1	1	1	1	1	1
Anisometropia	13		12		6	
Muscular Imbalance qt:	20′	R.D.*	20′	R.D.	20′	R.D.
Exophoria of 6 p.d. or more	4	33	8	31	11	22
Esophoria of 6 p.d. or more	3	4	6	9	9	6
Hyperphoria of 1 p.d. or more	1	0	1	0	0	0
Strabismus.						
Convergent	4		2		—	
Divergent	1		1		—	
Oblique	0		0		—	
Fusion deficiency	22		15		18	
Lateral dominance						
Eyedness						
Right	58		69		61	
Left	36		25		38	
Ambi	6		6		1	
Handedness						
Right	79		83		88	
Left	18		14		12	
Ambi	3		3		0	
Speed of recognition						
Below Q_1 of unselected cases, i.e., retarded						
Word recognition	49		17		25	
Object recognition	22		18		25	
Intelligence quotient below 90	24		11		6	

* R. D. = reading distance.

On the other hand, right handedness was less frequent among the poor readers, slightly more so in the ophthalmic group, and most frequent in the unselected classification. Left handedness occurred most often among the poor readers, least often among the unselected, while the incidence in the ophthalmic group fell between the others. Ambidextrousness was equally frequent among the poor readers and ophthalmic cases, but it failed to appear in the unselected group. Thirty-two percent more poor readers than ophthalmic cases, and 24 percent more poor readers than unselected cases exhibited significant degrees of retarded speed of word recognition. Important retardation in the speed of object recognition occurred most frequently among the unselected, slightly less so among the poor readers, and still less so among the ophthalmic cases.

The measure of central tendency used in this study was the median. The ophthalmic group presented a slightly lower median visual acuity than the other two groups in which the medians were equal and at the normal level. The median amblyopia was the same in all groups.

The unselected and ophthalmic groups presented equal medians for hypermetropia while the other group exhibited a 0.25D.-less median hypermetropia. The median myopia was approximately equal in all groups. Hypermetropic astigmatism occurred to a greater median degree in the ophthalmic

TABLE 2

CENTRAL TENDENCY OF EYE CONDITIONS EXHIBITED IN READING FAILURE, OPHTHALMIC, AND UNSELECTED GROUPS

Condition		Median of Reading Failure Group	Median of Ophthalmic Group	Median of Unselected Group
Age		9 yrs., 8 mo.	11 yrs., 6 mo.	10 yrs., 8 mo.
Visual acuity (Snellen)	O.D.	20/20	20/30	20/20
	O.S.	20/20	20/30	20/20
Amblyopia	O.D.	20/50	20/50	—
	O.S.	20/50	20/50	—
Refraction				
Hypermetropia	O.D.	0.75D.	1.00D.	1.00D
	O.S.	0.75	1.00	1.00
Myopia	O.D.	1.00	1.00	1.00
	O.S.	1.00	1.00	1.25
Hypermetropic astigmatism	O.D.	0.62	1.00	0.62
	O.S.	0.62	1.25	0.62
Myopic Astigmatism	O.D.	0.50	0.62	2.00
	O.S.	0.50	0.50	1.25
Muscular Imbalance				
Exophoria	20 ft.	4P.D.	4P.D.	3P.D.
	R.D.*	6	6	3
Esophoria	20 ft.	3	4	2
	R.D.*	4	6	2
Speed of recognition				
Object		0.0033 sec.	0.0100 sec.	0.0033 sec.
Word		0.0100	0.0050	0.0033
Intelligence quotient		102	103	109

* R.D. = habitual reading distance.

group as one might expect, while the other two classes presented equal and somewhat lower medians. The median myopic astigmatism was markedly higher in the unselected group, while the medians in the two other groups were considerably lower and very close to one another. In distant vision, the median exophoria was very close in all groups, but in near vision the reading failures and the ophthalmic cases presented medians that were twice as great as that of the unselected group. The median esophoria in distant vision was highest in the ophthalmic group, lowest in the unselected group, with that of the poor readers exactly between the others.

The median speed of word recognition was lowest among the reading failures and highest among the unselected, while the median speed of object recognition was equal and at the upper level in the reading failure and unselected groups but at a considerably slower level in the ophthalmic group. The latter exhibited a faster median speed of word recognition than of object recognition, suggesting that many of these people may have recognized the words by minimal cues without seeing them discriminatingly. The faster median speed of object recognition than of word recognition among the poor readers, on the other hand, suggests that language difficulty and unfamiliarity with word form may have influenced the scores of these pupils.

A difference of 10 percent in frequency was regarded as significant. Applying this more or less arbitrary standard it was found that the poor readers exhibited more frequent hypermetropia of one diopter or more, more exophoria of 6 prism diopters or more at the reading distance, greater retardation in the speed of word recognition, and more frequent intelligence quotients below 90 than did the unselected. This group also displayed more frequent left eyedness, more retarded speed of word recognition, and more intelligence quotients below 90 than did the ophthalmic group. As would be expected, the latter group exhibited a greater frequency of low vision than either of the others and a higher incidence of hypermetropia of one diopter or more, of anisometropia, and of exophoria at the reading distance than did the unselected group, being closer to the poor readers in these areas.

The differences between the medians of the various measurements in the three groups were not great. The ophthalmic cases presented a slightly lower median visual acuity than either of the others as might be expected. The difference in the medians of the various refractive conditions were less than one diopter except in myopic astigmatism, where the unselected group presented a somewhat higher median. The difference between the medians of the various phoria measurements ranged up to 3 prism diopters. It is noteworthy, however, that the poor-reading and ophthalmic groups presented medians of 6 prism diopters of exophoria at the reading distance as against 3 prism diopters among the unselected cases. This, as well as the higher frequency of the condition at the reading distance in the same groups, supports earlier findings.[3, 4]

The difference in medians of the speed of object and word recognition in the three groups ran about as one would expect, the medians for the poor readers and the unselected children being equal and high for object recognition, while that of the ophthalmic group was slower. The median speed of word recognition was slowest among the poor readers, faster but still retarded among the ophthalmic cases, and at a satisfactory level in the unselected group. The differences in median intelligence quotient in the three groups were too small to be important.

Conclusions

This study indicates that, for the present group at least, hypermetropia, exophoria at the reading distance, retarded speed of word recognition, and intelligence quotients below 90 occur more frequently among poor readers than in the other groups tested but

that the median amount of defectiveness is not appreciably greater in any of the groups. This is in line with the clinical observation that reading failures are often troubled to a greater extent than others by such handicaps as low degrees of hypermetropia and other eye defects.

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SLEEP WITH HALF-OPEN EYES (PHYSIOLOGIC LAGOPHTHALMUS)

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I spoke once in Shanghai with a Chinese colleague about my paper in which I described the diminished resistance of the cornea due to general disturbances of nutrition (Am. J. Ophth., **30**: 721-727 (June) 1947.) and to intoxication, and we spoke about the bad consequences of a slight lagophthalmus, when the general condition of the patient is poor. Prof. C. H. Chou then told me that many people in China sleep with half-open eyes and that his son has his palpebral fissure open from 3 to 4 mm. and that the cornea is also exposed. The globe sometimes turns around during sleep. Prof. C. C. Ho, head of the eye department of the Red Cross Hospital, who was listening to our conversation, declared with a smile that his lids did not close during sleep but that his cornea was not visible.

Even in the old Chinese history we find a narrative of the physiologic lagophthalmus of the Chinese.

In the time of the three kingdoms (230-264 A.D.) there lived a general, Chang Fei, whose habit was to keep his eyes open when he was fast asleep.

One day, he ordered a group of tailors to make 3,000 suits of uniforms in mourning fashion for his troops on account of the death of his "sworn brother"—the famous general Kwan Yu. This arduous task was assigned to be finished within a limited time of three days.

It was physically impossible to execute the order within such a short space of time, but General Chang insisted that it must be done or, failing this, all the tailors would be put to death. The tailors saw that death was inevitable and became desperate. They took council together and decided to assassinate the general and bring his head to the enemy, as the only way to save their own lives.

During the next night, two tailors made an attempt on the general's life. When they sneaked into his bedroom, to their great surprise, they found the general in his bed, rolling his widely opened eyes. They beat a hasty retreat, thinking that he was awake. A few minutes later they heard the general snoring loudly. The assassins entered the general's room again and purposely made some noise in order to see whether he was asleep or not. As he was not disturbed by the entry of the assassins, the two tailors stabbed the general several times, with fatal result.

The turning of the globe and cornea described in this story is the same as that seen in the son of Professor Chou, who moves his eyes, apparently to moisten the cornea and thus to hinder its desiccation.

In Nanking, I found that the wife of Dr. Chang, one of the assistants of the eye department of the Central Hospital, does not close her eyes entirely during sleep; apparently this happens not only in deep sleep during the night, but also when she takes a nap after lunch. The eye fissure during sleep is

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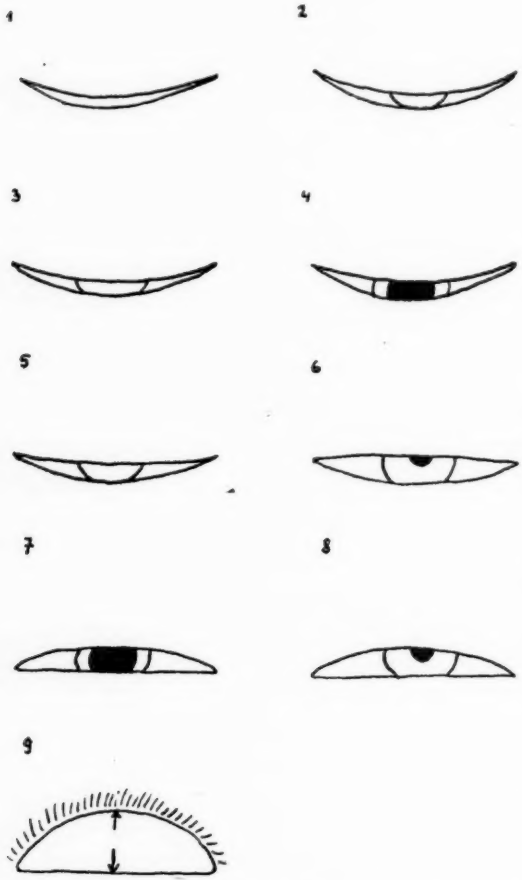
2- to 3-mm. wide and shows the lower third of the cornea.

Since this phenomenon appeared to be rather frequent I asked Dr. F. C. Wu, assistant of the Ophthalmological Department of the Shanghai Army Hospital to examine during the night a large group of students who live in the Army Medical College.

Examination of students

Dr. F. C. Wu. I examined the palpebral fissures of 500 healthy young Chinese students of the Army Medical College (aged 20 to 30 years) during their sleep. They were free of any pathologic factor which could affect the closure of the lids, such as scars of the lid, facial paralysis, thyrotoxicosis, and so forth.



Figs. 1 to 9 (Fuchs and Wu). Appearance of various degrees of lagophthalmos is shown in Figures 1 to 8. Diagram of tarsus height is shown in Figure 9.

Of these 500 cases 23 (4.6 percent) had their lids open 1 to 2 mm. during sleep and two cases (0.4 percent) had their lids open 3 to 4 mm. during sleep.

In the first group four different positions of the cornea were observed: (1) In 4 cases the cornea was not visible (fig. 1). (2) In 10 cases the lower margin of the cornea just touched the border of the lower lid (fig. 2). (3) In 3 cases about 1 mm. of the cornea was covered by the lower lid (fig. 3). (4) In 6 cases the central part of the cornea and parts of the pupil were not covered and were visible in the palpebral fissure (fig. 4).

As these cases show, a considerable number of persons with lagophthalmus do not show Bell's phenomenon during their sleep. The pupil is apparently not always narrow, as physiologic textbooks would lead one to believe.

In the second group of two students with palpebral fissures opened 3 to 4 mm. during sleep, the first student, aged 25 years, revealed an open fissure of 3 mm. on the right. The lower edge of the cornea touched the border of the lower lid (fig. 5). The left eye was exposed 4 mm. during sleep, the lower part of the pupil was visible, and the lowest millimeter of the cornea was covered by the lower lid (fig. 6).

The second student, 23 years of age, had both eyes open to an extent of 3 mm. The middle of the cornea and pupil were visible; about 2 mm. of the lower cornea was covered by the lower lid (fig. 7).

Later on, a third student, aged 20 years, was found to have a lagophthalmus of 3 mm. in both eyes during sleep. A small piece of the pupil was visible and about 1 mm. of the lower cornea was hidden by the lower lid (fig. 8).

Comment

Dr. A. Fuchs. From the report of Dr. Wu and the three eye doctors, Professor Chou,

Professor Ho, and Dr. Chang, it is clear that, in a considerable number of cases, the lids are not closed during sleep, a condition of which I have not heard anything in Europe. The frequency is emphasized when in addition to the statistics of Dr. Wu, it is known that three ophthalmologists have the condition themselves or in a member of their families.

Two possible causes of this physiologic lagophthalmus have to be considered: (1) a shortness of the upperlid, as is seen in cases of congenital familial ptosis. Nothing of this nature could be recognized in the eyes of the eye doctor and the son. It is also certain that these people close their eyes in a normal way during day.

A second possibility would be that (2) the facial nerve does not have a sufficient and constant effect on the musculus orbicularis. It is known that in unconscious persons, the relaxed middle position of the lids leaves part of the palpebral fissure open. During sleep, a special tonus or spasm of the orbicularis must be present, just as a tonus of the sphincter of the rectum and of the urinary bladder is physiologically present.

TABLE 1

HEIGHT OF UPPER EYELID IN 100 CHINESE

Sex	No.	Age	Eyelid Height		
		(Aver.)	Max.	Aver.	Min.
Female	10	25.6 yrs.	9.5 mm.	8.25 mm.	6 mm.
Male	90	26 yrs.	9.5 mm.	7.95 mm.	6 mm.

ROLE OF TRACHOMA SCARS

After examining several hundred cases of trachoma in China, I was astonished to find that the characteristic scars of trachoma in the sulcus subtarsalis, usually illustrated in European textbooks, are by no means common in China. More frequent is another type of trachoma scars which come from the upper edge of the tarsus and from the fornix and radiate like flames into the tarsal conjunctiva. The following possible causes of this type of trachoma scars suggested themselves:

1. That the blood vessels, which perforate the middle part of the tarsus and supply the tarsal conjunctiva, are not present in the lids of the Chinese. One assumes that the infiltration of the submucosa advances along these vessels into the tarsus and so causes the typical scars in the sulcus subtarsalis.

Since an anatomic examination of the lids was not possible, I was not able to pursue this probability.

2. Possibly the height (fig. 9) of the tarsus is much smaller in China than in Europe. If the tarsus is less high, it would be easy to understand the absence of the scar formation in the sulcus subtarsalis and the boat-shaped distortion of the tarsus so characteristic of trachoma in Poland, Russia, and the Near East.

Since the shortness of the tarsus could play a considerable rôle in physiologic lagophthalmus, I begged Dr. Wu to examine a number of students and measure the height of the tarsus.

TABLE 2

VARIATIONS IN HEIGHT OF UPPER TARSUS

Height	Percent of Subjects
6 mm.	3
6.5 mm.	2
7 mm.	11
7.5 mm.	20
8 mm.	31
8.5 mm.	16
9 mm.	13
9.5 mm.	4

Examination of tarsus

Dr. F. C. Wu. The height of the upper eyelid in the Chinese (fig. 9) was examined in 100 normal persons—90 males and 10 females. The results are shown in Tables 1 and 2.

A tarsus of 6 mm. was present in two men and one woman. A 9.5-mm. tarsus was present in two men and two women. The average height of the upper tarsus in Chinese is apparently between 7.5 and 8.5 mm. The incidence of high tarsus is apparently greater in women than in men.

In three cases of lagophthalmus during sleep, in which the patients showed an opening of 3 mm. or more, the upper tarsus measured 8.5 mm. in height in two cases and 9.5 mm. in one case.

Conclusion

Dr. A. Fuchs. Apparently the upper tarsus in the Chinese is less high than the tarsus of European people. Examination of 10 Europeans residing in China showed only one 7.5-mm. measurement and several of 9.5 mm. and 10 mm.

Certainly this little difference in height of the upper tarsus cannot be the cause of the physiologic lagophthalmus of the Chinese, since the upper tarsus of the three persons with open palpebral fissures of 3 mm. and more had a tarsus height which was maximum.

From this it would seem that the second supposition—that the tonus of the orbicularis is weakened during sleep—is more probable. Since the cornea is exposed, it is interesting to conjecture why the cornea does not become desiccated. The cause may be that these globes, when the Bell's phenomenon is not active, are rolling during sleep and so moisten the cornea. The story of the Chinese general, Chang Fei, and the observations of Professor Chou, who reported that his son makes slow, rotating movements of the eyes during sleep, would seem to substantiate this.

INTRAOCULAR PRESSURE OF NORMAL AND GLAUCOMATOUS EYES AS AFFECTED BY ACCESSORY LIGHT STIMULI*

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Moscow, U.S.S.R.

The influence of accessory stimuli upon the visual functions has now been safely established. On the basis of the data available it can be assumed that accessory stimuli also affect such an objectively recordable function of the eye as the intraocular pressure.

The present investigation is concerned with the effect of a special accessory light stimulus in the form of illumination of the second eye from a white or colored light source.

I. EXPERIMENTS WITH WHITE LIGHT

METHODS

The patient, reclining on his back, was allowed to adapt himself for 10 minutes to the illumination of the room in which the experiment was to be made. Then, the intraocular pressure of his right eye was measured with a Schiøtz tonometer after it had been anesthetized with a 1-percent dicaine solution.

The light was then switched off, and the patient was left in an absolutely dark room for one hour. After this, one eye of the patient was covered with a black eye shield, and the other eye was illuminated for 30 minutes by means of a special device arranged in the following manner. A cone-shaped tube 40 cm. in length, was suspended over the left eye of the patient. The tube contained at its base a 75-watt frosted bulb. The brightness of the lightest spot of the bulb was equal to about 16 foot-candles. The left eye of the observer had to be fixed on this spot. The illuminating cone had several lateral apertures which reduced the possible effect of temperature.

During the time that the left eye was exposed to the light stimulus, the patient's right eye was repeatedly subjected to tension measurements at approximately 5-minute intervals. The tension of this eye was also measured some 15 to 20 minutes after the elimination of the accessory stimulus; that is, under the initial illumination conditions of the laboratory room.

The experiments were carried out on 35 subjects, 14 of whom had normal eyes, and 21, glaucomatous eyes. The investigative procedure just described was applied to both normal and glaucomatous subjects. The glaucoma subjects were divided into two groups. One group comprised cases of clearly pronounced glaucoma. In the second group were patients displaying prodromal glaucoma, as well as the "normal" eyes of glaucomatous patients.

RESULTS

The results obtained demonstrated that, under the influence of unilateral illumination of one eye, the intraocular pressure of the other eye was reduced. Our experiments likewise showed that the variation in the tension of glaucomatous eyes exposed to light or to dark follows the same general course as that revealed by our experiments on normal eyes.

In glaucomatous eyes, however, the reduction in the intraocular pressure is more sharply pronounced, as a rule. A certain smoothness of the curve, typical of a normal eye, vanishes, giving place to a curve which is not infrequently distinguished by its broken shape.

Typical curves illustrating the drop of intraocular pressure resulting from illumination of the second eye both in normal and glaucomatous observers, are plotted in Figure 1, A and B.

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The pupillary reflex does not seem to have any appreciable bearing upon the variation in the intraocular pressure, when the second eye is exposed to light. From 16 experiments carried out upon 12 patients whose pupillary reflexes were eliminated by means of homatropinization, it may be seen that under these conditions the tension of the tested eye is likewise reduced by illuminating the second eye (fig. 2).

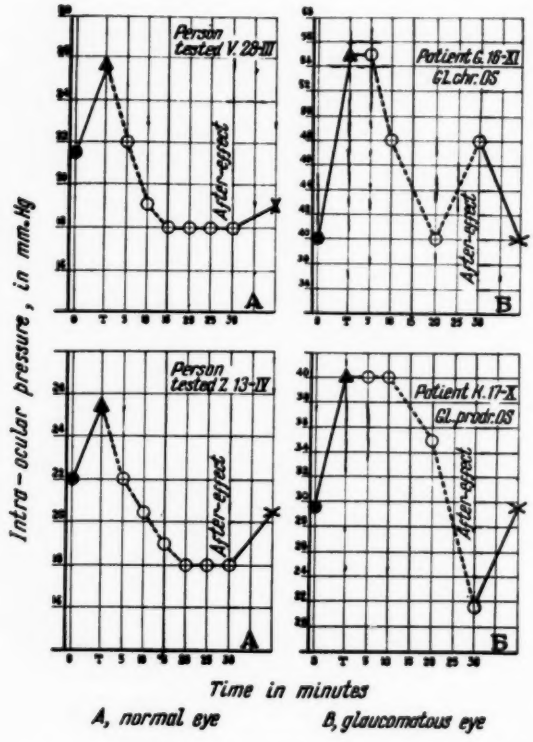

Fig. 1 (Zaretskaya). Intraocular pressure of one eye as affected by illumination of the other eye. Along the ordinate are plotted the values of the intraocular pressure in mm. Hg; along the abscissa, time in minutes. Black circles denote the initial tension found under general illumination of the experiment room; black triangles stand for points corresponding to the level of tension measured after dark adaptation of the eye for one hour; open circles and broken line denote data obtained by illuminating the second eye for 30 minutes; a solid line with crosses shows values obtained under initial light conditions, 15 to 20 minutes after exposure has been stopped.

The reduction of tension is dependent on the brightness of light acting upon the second eye. Thus, by exposing one eye to a light intensity of 3.3 or 0.28 foot-candles, we obtained, in an average of 26 tests, a maximum drop of tension in the second eye of 2.7, 5, and 9 mm. Hg, respectively. The tests also showed that an increase in the brightness of the light stimulus is accompanied by an increase in the rate of reduction of intraocular pressure in the second eye.

Discussion

The mechanism underlying the influence of dark or light upon intraocular pressure still remains obscure. But the material available does not seem to corroborate the purely mechanical interpretation given this phenomena by certain authors, who ascribe the main role in the changes of tension to the pupil reaction (Grönholm, Seidel).

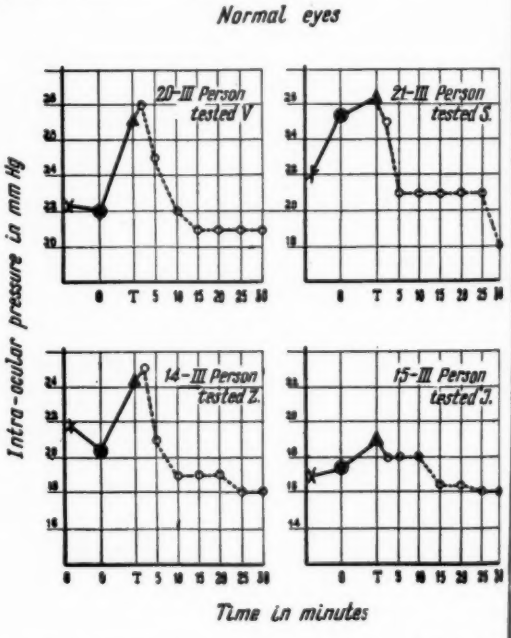

Fig. 2 (Zaretskaya). Intraocular pressure of the right eye as affected by illumination of the left eye. (Homatropine test.) Along the abscissa is plotted time in minutes; along the ordinate, intraocular pressure in mm. Hg. A cross denotes intraocular pressure at the start of measurement; a black circle, tension measured upon instillation of homatropine; a triangle, data obtained after one hour of dark adaptation; open circles and broken line, tension in the illuminated eye.

Feigenbaum's interpretation; namely, that the vascular system of the eye is affected by light through the vasomotors appears more plausible. The action of light may in this case be effectuated through the pituitary body and the vegetative centers whose excitation results in vasomotor reactions involving the optic apparatus, as well.

What are then the decisive conditions responsible in a glaucomatous eye for an increase in its tension, when kept in the dark, and for a reduced tension, when exposed to light?

Most of the authors look for an answer in a definite state of the neurovascular system of a glaucomatous eye; that is, in the so-called neurovascular reflex of Kalfa. This problem has not yet received any definite or satisfactory solution.

II. Experiments with color illumination

Thanks to the experiments carried out by Prof. S. V. Kravkov, it has been established that the green- and red-sensitive apparatus of our vision responds in an opposite way to such vegetative drugs as adrenalin and pilocarpine. It seemed, therefore, consistent to anticipate that the vegetative reactions of the organism will also be different in the case of the eye exposed to red or green stimulants.

Method

The procedure adopted was similar to that employed in our experiments with white light herein described.

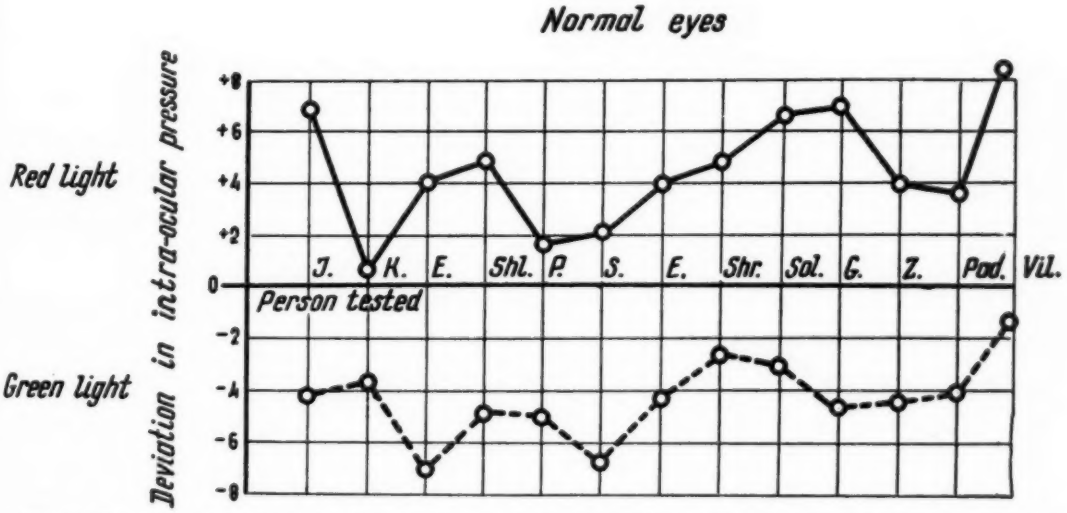

Fig. 3 (Zaretskaya). Effect of light stimulation of the one eye by colored light upon intraocular pressure of the other eye. Along the ordinate is plotted the maximum change in the intraocular pressure (in mm. Hg) from the level of dark adaptation. Along the abscissa are plotted the different persons tested. Circles and the solid line denote changes in intraocular pressure obtained by stimulating the other eye with red light; circles and the broken line, the results for the green light.

A 150-watt incandescent lamp served as a source of light stimuli. It was placed in a special pasteboard cone 40-cm. long, adjusted to the eye of the person tested, and supplied either with a green or a red gelatine filter. The green filter was transparent for rays approximately within the range of 433 to 586 mμ; the red filter for 578 to 720 mμ. Both the red and green light were equal in brightness.

The eye of each observer was exposed for 30 minutes, one day to red light, another day to green light. The experiments were carried out on 17 persons with normal eyes and on 25 glaucomatous patients.

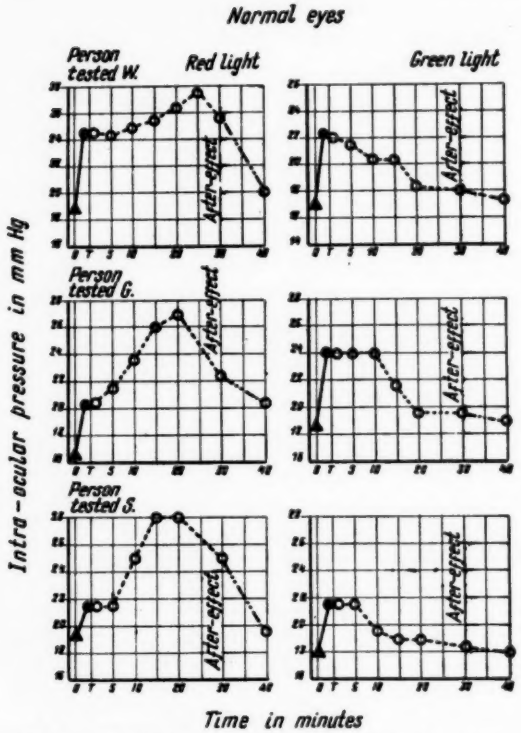

Fig. 4 (Zaretskaya). Curves of intraocular pressure (red or green light applied to the other eye). Along the ordinate are plotted the values of intraocular pressure in mm. Hg; along the abscissa, time in minutes. Triangles denote the initial pressure measured under general illumination of the room; black circles show the level of pressure with dark adaptation of the eye; open circles and broken line are readings obtained when the second eye was exposed either to red or green light; circles and dotted line show tension values found after stopping exposure of the second eye to the light stimulus (that is under the initial illumination conditions).

Results

The experiments with normal subjects showed the reaction of the intraocular pressure to be a reverse one, according to whether red or green light was employed.

The exposure of the second eye to green light brought a reduction of the intraocular pressure of the investigated eye; with the red light, the effect was reversed (fig. 3).

A similar picture of a reverse reaction to the red or green light is offered by Figure 4, which shows individual curves plotted for several patients tested by us.

As borne out by Figure 4, the curves obtained with red or green light show a different run.

Comment

Glaucomatous eyes display a well-pronounced specific reaction of their intraocular pressure for the same color stimuli. Thus, in all experiments carried on by us an appreciable reduction in tension was observed when the second eye of the subject under test was exposed to green light; the hypotonic effect in experiments with glaucomatous eyes was much stronger, however, than in experiments with normal eyes.

The hypotonic effect of green light appears still more conspicuous if compared to the effect of white light even of a higher brightness applied to the same glaucomatous patients (6 subjects) for sake of experimental control.

Table 1, as well as Figure 5, substantiate this observation.

Patients with a sharply pronounced form of glaucoma display a conspicuous deviation

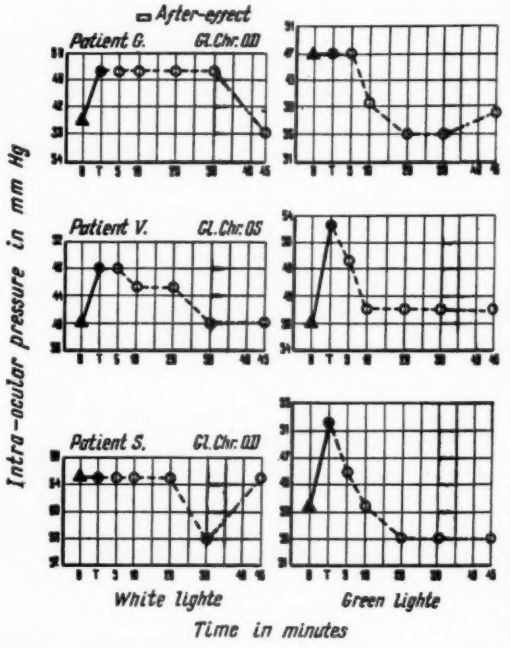

Fig. 5 (Zaretskaya). Intraocular pressure of one eye when the other eye is illuminated with green or white light. (For chart details, please see legend of Figure 4.)

TABLE 1

INTRAOCULAR PRESSURE IN GLAUCOMATOUS EYES AS AFFECTED BY WHITE OR GREEN LIGHT STIMULI

No.	Patient	Diagnosis	Tension Deviation in mm. Hg. Under Exposure of the Other Eye to	
			White Light	Green Light
1	F	Glaucoma chron.	−5,0	−10,5
2	S	" "	−6.0	− 5.5
3	G	" "	0	−12,5
4	V	" "	−8.0	−13.0
5	R	Glaucoma absol.	0	− 8.5
6	Sa	" "	0	−17.0

from the normal in the reaction of their intraocular pressure to red light. In normal subjects, however, the stimulation of the one eye with red light almost always results in a rise of the intraocular pressure of the second eye; no increase of intraocular pressure has, as a rule, been observed in glaucomatous eyes.

The intraocular pressure of the glaucomatous eye either remains stable, or may even show a slight tendency to decrease (fig. 6).

A similar picture of different responses to the red and the green light may be seen also in Figures 7 and 8, representing individual curves bearing out the results of experiments carried out on definite days.

Discussion

The reaction of intraocular pressure in response to red light, which is missing in glaucomatous patients, points to a definite disturbance between their color-perceiving apparatus and their autonomous nervous system. The green-perceiving apparatus of these patients may possibly be more active than in normal subjects.

It is of interest to recall the remark made by Khodin as early as 1897 (*Journal for War Medicine*, 1897, in Russian), namely, that the increase in intraocular pressure is accompanied with a definite shift of color perception toward the green.

Wessely (1927) has described the presence of scotoma for the red as a symptom of glaucoma.

Some changes in color sensitivity have also been described in glaucomatous patients by Colomba (1932), who is likewise inclined to regard the analysis of color per-

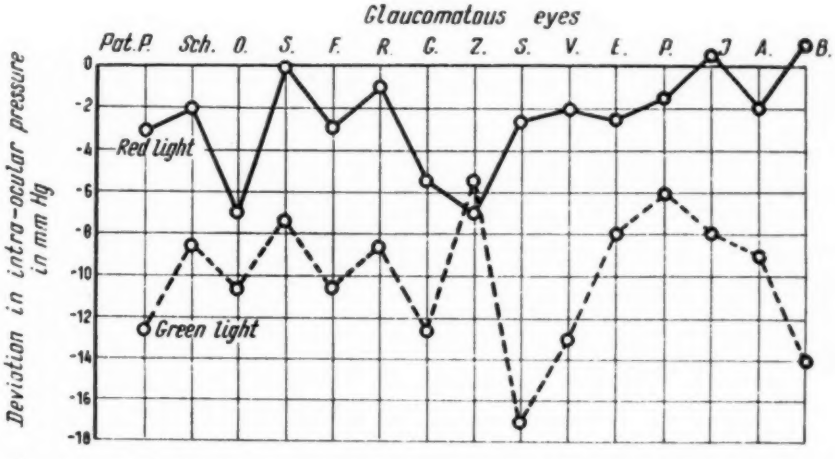

Fig. 6 (Zaretskaya). Intraocular pressure of one eye as effected when the second eye is exposed to colored light. (For chart details, please see legend of Figure 3.)

ception as one of the tests helpful in an early diagnosis of glaucoma.

Pointing out the importance of the investigation of color-vision disturbances for the diagnosis of glaucoma, Oloff (1932) remarks that they are of rather frequent occurrence in subjects affected by this disease.

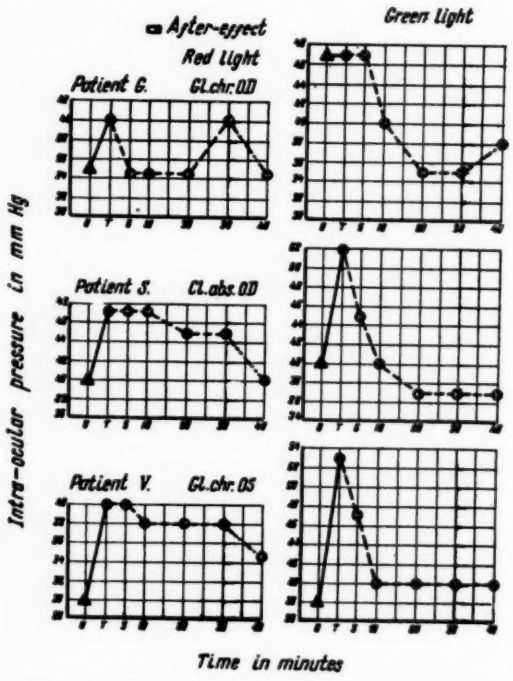

Fig. 7 (Zaretskaya). Change in intraocular pressure produced in one eye of glaucomatous patients by illuminating the other eye with red or green light. (For chart details, please see legend of Figure 4.)

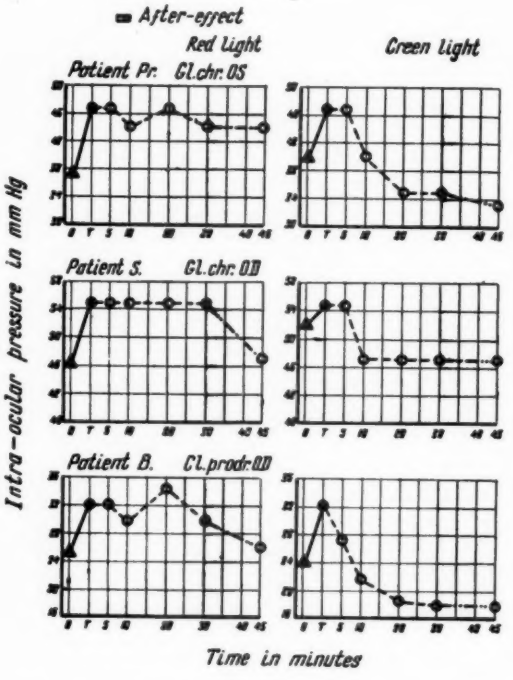

Fig. 8 (Zaretskaya). Change in intraocular pressure produced in one eye of glaucomatous patients by illuminating the other eye with red or green light. (For chart details, please see legend of Figure 4.)

Conclusions

Without claiming to have deciphered the mechanism of action of the accessory stimuli applied by us upon the intraocular pressure in a normal or a glaucomatous eye, the author, nevertheless, feels justified in advancing the following conclusions based upon her experiments.

1. The lowering intraocular pressure caused by vegetative reactions due to accessory stimuli is less in normal eyes and proceeds in a more uniform way, along a smoother curve, than in eyes of glaucomatous patients.

2. Fluctuations in intraocular pressure caused by darkness and light are likewise much more sharply expressed in glaucomatous eyes than in normal ones.

3. The pupillary reflex produced no appreciable effect upon the change in intraocular pressure when the second eye was exposed to the light stimulus.

4. The reduction in the intraocular pressure of the eye tested is dependent upon the brightness of the light stimulus applied to the second eye.

5. The opposite nature of the change in intraocular pressure, as brought about by the green or the red light stimuli, is evidence of the fact that the excitations of the green-perceiving apparatus of vision and of its red-perceiving apparatus produce two an-

tagonistic reactions of the autonomous nervous system.

6. The fact that, in contrast to unaffected subjects, glaucomatous patients do not show the usual increase in intraocular pressure in response to red stimuli of the eye, gives us ground to believe that the determination of the response of the intraocular pressure to the red light may become a supplementary method for diagnosing glaucoma.

7. In so far as stimulation with green light is followed by a particularly marked drop in intraocular pressure in glaucomatous patients, there seems to be some hope that green light may be applied to such patients for therapeutic purposes in the form of green spectacles used for reducing their intraocular pressure.

Sadovaya-Chernogryazskaya Street (14/19).

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NOTES, CASES, INSTRUMENTS

A SIMPLIFIED TANGENT-SCREEN TECHNIQUE USING A PANTOGRAPH ATTACHMENT

ADOLPH POSNER, M.D.
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The pantograph attachment shown in the accompanying figure was designed for the purpose of transferring the field from the tangent screen onto a chart. When employed in conjunction with the technique to be described, it offers several advantages over the methods in general use.

1. It reduces the time consumed in tangent-screen examinations and eliminates the necessity for freehand drawing by making the procedure of transferring the field quick and automatic.

2. It enhances the accuracy of the field chart and thus makes possible more careful comparative field studies, so essential in the evaluation of the course of a case of glaucoma.[1]

3. It permits the use of an unmarked screen, so that the examiner is not influenced by the outline of the normal blind spot or by the other markings.

4. It makes it possible to relegate to an untrained assistant the task of charting the field.

5. It enables the perimetrist to use the same screen at any desired distance by selecting the chart appropriate for that distance.

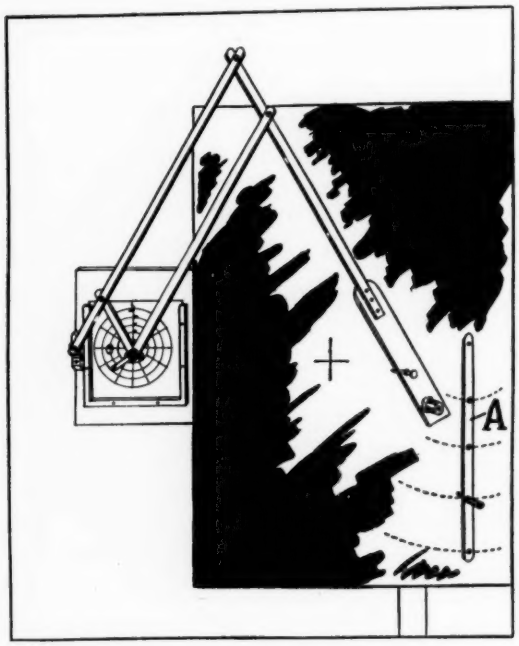

Fig. 1 (Posner). *Pantograph attachment for tangent screen.* This device is used for transferring the field automatically onto a chart. It has a reduction ratio of 1:8. The end segment is made of a transparent plastic. The tangent screen is left unmarked. A wooden rod (A) may be used for drawing temporary markings on the screen. Perforations placed at 5-degree intervals accommodate a wax crayon. The rod thus serves both as a ruler and compass.

The pantograph is a device widely used by artists and draftsmen for reducing and enlarging drawings accurately and automatically. Its use in connection with the tangent screen was suggested by Downey[2] and by Marks.[3] The devices described by them failed to gain popularity, probably because they were intended for automatic registration, rather than for copying the field, and thus could not help but be too cumbersome and obtrusive to be practical.

The pantograph attachment designed for use with the tangent screen reduces the field to one eighth its actual size. If a screen, one-meter square, is used, the corresponding field on the chart would be five-inches square. The writing arm is provided with a bushing which accommodates either a lead pencil or a colored crayon. The end section of the tracing arm is made of a transparent plastic, so that both the tracing style and the screen markings are under continuous observation. The frame which holds the chart is adjustable, so as to permit exact centering of the chart with reference to the screen. This adjustment also allows for a considerable range in screen sizes which

may be used with the same pantograph.

The technique of tangent screen perimetry, as herein briefly outlined, is well suited for use in conjunction with the pantograph attachment. The tangent screen is one-meter square and is made of black wool felt stretched over a wooden frame. A wooden disc, six inches in diameter, is attached to the frame behind the screen in such a way as to serve as a support for the central part of the screen. The screen does not carry any markings and is thus adaptable for use at other distances. The subject is seated so that his eyes are at one-meter distance from the center of the screen and on the same level. Uniform, shadowless illumination of about 5 to 7 foot-candles is provided by a Circline fluorescent tube mounted on a floor stand which carries an adjustable chin rest for head fixation. This accessory unit will be more fully described elsewhere.

The test objects which were found most useful are the white spherical ones devised by Evans. They range in size from 0.4 to 1.5 mm. The set* also contains larger disc-shaped test objects which may be used either as white or as colored stimuli by coating them with chalk of the desired color.

The field is plotted on the screen with colored chalk, using different colors to represent different sizes of stimuli and different eyes. A simple notation, made in the corner of the screen with each color used, serves as a key for the identification of the lines in the subsequent charting of the field. For example: "R-1" written in the blue chalk means that the blue line represents the field of the right eye as plotted with a 1-mm. white test-object. The colored chalks should be dry, nonwaxy, and of dark shades.† The chalk marks may be removed with a soft brush. Occasionally the screen should be cleaned with a vacuum cleaner. A felt screen is preferable to a woven one, since the chalk dust remains close to the surface.

The method of plotting the field is an adaptation of that employed by Evans for angioscotometry. For a detailed description the reader is referred to Evans' excellent monograph.[4] One fundamental principle might bear emphasizing here: *The stimulus should always be moved perpendicularly to the border of the scotoma.* This presupposes a knowledge of the shapes and locations of the typical scotomas for each condition, together with the most common variants.

Tangent-screen perimetry is an integral part of the ophthalmologic examination and, as such, should preferably be carried out by the ophthalmologist himself, if the findings are to have any clinical significance. Good perimetry requires special study and experience, which can be gained only by careful work and by correlating the field with the clinical picture. It is hoped that the pantograph attachment here described, by reducing the time consumed and by eliminating the need for free-hand drawing, will extend the use of the tangent screen in ophthalmologic practice.

667 Madison Avenue (21).

* Made by the National Electric Instrument Co., Long Island City, New York.

† Obtainable in most artists' supply stores.

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THE PREVENTION OF CYCLOPLEGIC GLAUCOMA

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Chicago, Illinois

"What precautions must be used in daily practice to assure adequate examinations of eyes at any age without danger of cycloplegic glaucoma?" was one of the questions asked in the American Academy's Home Study Course on Refraction. In spite of the obvious importance of the question, the answers given by the students were, almost without exception, very unsatisfactory. This communication has two aims, therefore: (1) to find out why the answers were so poor, and (2) to focus attention of all prospective students on a correct approach to the problem. It must be remembered that the home study course stresses the academic approach with particular emphasis on the basic sciences; and, as a consequence, the reading which is required of the students is the standard accepted textbooks on any given subject.

REVIEW OF REFERENCES

Review of these references for the subject of refraction showed that the standard textbooks left much to be desired in their discussion of the problem. Thorington,[1] Fuchs,[2] and Cowan[3] briefly mention in a scant sentence or two that cycloplegics should not be used in the presence of glaucoma. May[4] mentions the fact that the tension should be checked, but does not specify a tonometric tension. Duke-Elder[5] states that the presence of glaucoma should be excluded before using mydriatics, and that miotics should be instilled afterward. The *American Encyclopedia of Ophthalmology*[6] suggests taking the tension first, and also says it is "a good plan" to instill a miotic afterward. O'Rourke in Berens[7] says that caution is indicated, and that a cycloplegic should not be used until a fundus examination is made and the tension tested digitally, and, if there is any doubt, tonometrically. Gifford,[8] although brief, gave the most complete answer when he said that the intraocular pressure should be estimated in all people over 25 years of age, the fundus observed through undilated pupils to rule out the presence of a glaucomatous cupping, and a miotic instilled in all people over the age of 20 years, at the conclusion of the examination.

Discussions in these same textbooks under the heading of glaucoma were no more complete in so far as this particular question was concerned. Here the problems of diagnosis were discussed with regard to provocative tests, perimetry, and tension curves in early stages. Such procedures obviously are extremely important in confirming a suspected case, but they can hardly be carried out as routine on every case for cycloplegic refraction.

PRECAUTIONS TO OBSERVE

What then are the precautions to be observed? What steps must be taken to prevent a cycloplegic glaucoma? First, it is important when the history is being taken to ask specifically whether the patient has ever seen halos around lights, whether there is any history of glaucoma in the family, or whether the patient has ever had any episodes of ocular inflammation suggestive of attacks of acute glaucoma. It goes without saying that the visual acuity must be determined, and in those cases where it is possible a manifest refraction should be performed.

The next step is to make an external examination of the eyes, paying particular attention to the size of the cornea, the depth of the anterior chamber, the size of the pupils, and the pupillary reactions. Any deviation from the normal in the way of a small cornea, a shallow anterior chamber, or pupils which seem unduly large and do not react to light briskly should arouse suspicion and require that further study be made to rule out glaucoma. A careful ophthalmoscopic inspection of the optic discs

must be made in every case, and any case in which there is any suspicion of a cupping, must have a complete study.

Clinical routine

It is a good clinical habit to check the tactile tension in every case, but no clinician can appreciate a difference of 5 mm. Hg by this technique. Therefore, it is extremely important to check the intraocular pressure *tonometrically* on all patients over 25 years of age before a cycloplegic is instilled. This measurement should be rechecked tonometrically after the pupils are dilated. Any case in which there is an increase of intraocular pressure of 5 mm. Hg or more, following dilatation of the pupils, is suspect and requires an immediate careful workup and prolonged follow-up. Gradle[9] has shown that in 2.8 percent of 500 eyes, there was an increase of 5 mm. Hg or more in the intraocular pressure following the use of cycloplegic for refraction.

Following the use of a mydriatic it is important to instill a miotic to hasten recovery from the effects of a dilated pupil in all cases. In those cases in whom there is an abnormal increase in intraocular pressure under mydriatic, it is absolutely necessary to keep the patient in the office or clinic until both the tension and pupils have returned to normal with the use of the miotic. Subsequent treatment is then decided on after a careful glaucoma work-up.

The importance of these procedures cannot be over-emphasized. The practice of routinely dilating all pupils without adequate examination is not only poor ophthalmology, it is dangerous, and it is important that all those who are responsible for the teaching of our young men recognize and emphasize this point.

58 East Washington Street (2).

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QUININE AMAUROSIS*

B. L. Braveman, M.D., D. S. Koransky, M.D., and M. M. Kulvin, M.D.
Hines, Illinois

Quinine in many instances is the drug of choice for the prophylactic and active treatment of malaria. With the return of many servicemen who have suffered from malaria and the easy availability of quinine at present, toxic reactions to the drug will probably be seen more frequently.

The toxic action of quinine in the retina is reported as being (1) directly toxic to the ganglion cells and (2) acting upon the retinal arterial system to produce extreme vasoconstriction with resultant ischemia and damage. Patients in most of the reported

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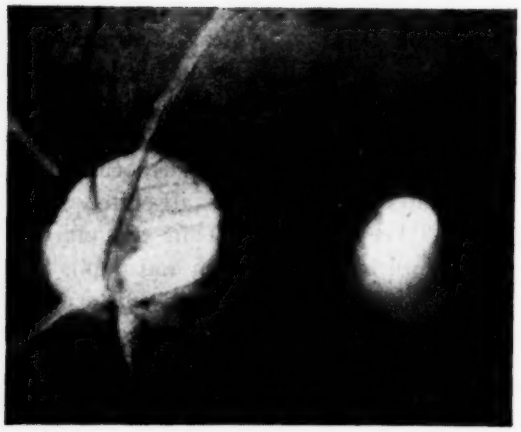

Fig. 1 (Braveman, Koransky, and Kulvin). Fundus picture in a case of quinine amaurosis four weeks after poisoning showing optic atrophy and silver-wire arteries.

cases recovered much of their central vision. However, varying amounts of peripheral constrictions remained. Duke-Elder states, "In all cases the tendency is for recovery: in the milder degrees of poisoning the vision may recover in a few hours but in more severe cases amaurosis may persist for several days or even weeks, and in this event some degree of atrophy of the nerve and some permanent loss of vision is inevitable. Permanent blindness, however, has not been recorded."

The following case is the report of an individual who ingested a large overdose of the drug and developed severe toxic cardiovascular and retinal damage.

Report of a case

A 42-year-old white man, a bartender, was admitted to the hospital on December 29, 1946, in a comatose condition. No history was obtainable.

On initial examination systolic blood pressure was 60, diastolic pressure could not be recorded. The pulse rate was not obtainable. An emergency electrocardiogram recorded a ventricular tachycardia. The patient was given immediate emergency management of oxygen, and so forth. The tachycardia and low blood pressure persisted for a period of eight hours. Gradually the blood pressure rose, the pulse rate fell, and the patient responded to treatment. Within 48 hours after admittance, the patient regained consciousness and at that time stated that the room was dark.

An ophthalmologic examination revealed light perception and projection to be absent in both eyes. The pupils were round, measured 4½ mm., and fixed to light and accommodation. Ocular movements, adnexa, and anterior segments were normal. Both fundi revealed well demarcated, flat discs. The retinas appeared markedly pale and somewhat edematous. The maculas were cherry red against the pale retinal background. The arterial tree was markedly attenuated, the arteriovenous ratio being approximately 1 to 4, and the arterial tree could only be followed out to the region of the equator.

Diagnosis. A provisional diagnosis of retinal ischemia was made and vasodilators were administered in large doses (intravenous sodium nitrite, papaverine, and niacin by mouth).

Course. Within 72 hours, the retinal edema receded and the fundi regained a normal color. However, the vascular tree remained constricted.

Seven days after the original eye examination, the arterial tree was completely sheathed and had the appearance of silver wire. The discs became progressively paler and were dead white in appearance by the second hospital week and have remained so.

Fifty days after admission faulty light perception and projection were obtained; shortly thereafter finger counting at 18 inches could be elicited. The pupils remained fixed to light and accommodation and field studies could not be obtained.

Five days after admission, a history of excessive doses of quinine was obtained from the patient and confirmed by his wife. He stated that it was his custom to take 45 grains of quinine daily when he felt an attack of malaria coming on. On December 28, 1946,

he took all of the quinine that he had available, which consisted of approximately 100 tablets varying in dose from 2 to 5 grains each. At that time the diagnosis of quinine intoxication was established. The patient did not complain of impaired hearing, but audiometric examinations revealed partial nerve deafness bilaterally.

Summary

This is a case of quinine poisoning with bilateral amaurosis persisting for a period of at least 50 days. The probable dose was at least 200 grains (Goodman and Gilman state that the fatal dose is 120 grains). The patient also suffered from a cardiovascular episode which consisted of a ventricular tachycardia which persisted for a period of eight hours and was associated with the marked fall in blood pressure. The outstanding findings in the fundi was the markedly persistent vasoconstriction that led to severe retinal damage.

Veterans Administration Hospital.

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Historical Miniature

Thomas Young, who first described astigmatism when he published measurements of his own eyes in 1801, was also the first to discover the very common occurrence of hypermetropia. The recognition of this fact, which Donders did not mention in his "Refraction and Accommodation," in 1866, is of far greater importance than the description of an isolated case of extreme hypermetropia which was published by Janin in 1772.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

May 5, 1947

DR. BENJAMIN FRIEDMAN, *president*

SURGICAL ANATOMY OF THE EYE

DR. RAYMOND E. MEEK discussed this subject during the instruction hour.

BIFOCAL SEGMENT SELECTOR FOR SPECTACLE FRAMES

DR. AARON ROTH demonstrated an attachment to spectacle frames. This can be incorporated in the frames, and allows the wearer to move his bifocal segment up so that he can use it directly in front of his eyes if necessary.

CENTRAL SEROUS RETINOPATHY

DR. EMANUEL ROSEN presented two cases of central serous retinopathy following vaccination. He demonstrated that the macula may be involved after vaccinia inoculation. Such an involvement usually occurs within an incubation period of 7 to 11 days. Histologically, it simulates the picture seen in the brain in cases of postvaccinial encephalitis. Dr. Rosen stated that we may assume this to be an allergic response with specific localization in the macula.

PERIPHERAL VISUAL ACUITY WITH SPECIAL REFERENCE TO SCOTOPIC ILLUMINATION

DR. JOSEPH MANDELBAUM'S paper (Louise L. Sloan collaborating) was published in the JOURNAL **30**: 581 (May) 1947.

In his concluding remarks Dr. Mandelbaum said that:

1. Scotopic peripheral acuity does not parallel the rod population or the light sensitivity of the retina.

2. Maximum scotopic acuity can be achieved by 4- to 8-degrees' eccentric fixation.

3. Light intensity is not as critical a factor in peripheral acuity as it is in central acuity.

4. At the lowest scotopic levels, visual acuity is fairly constant from 4-degrees to 30-degrees' eccentric fixation.

5. A considerable overlap occurs between cone and rod function, the paracentral rods dominating discriminatory function at light intensities well above the cone light threshold.

6. For 25- and 30-degrees' eccentric fixation, the rod cells dominate form discrimination even at intensity levels as high as one millilambert.

USE OF PLACENTAL EXTRACT IN VIRUS DISEASES OF THE CORNEA

DR. BERNARD KRONENBERG described the preparation of aqueous placental extract, lipoid extract, immune gamma globulin from placental blood, and placental tissue coagulant. He gave a preliminary report on the use of aqueous placental extract in the treatment of herpetic keratitis and superficial punctate keratitis. In view of the favorable results obtained in these cases, Dr. Kronenberg stressed the need for further clinical investigation.

Dr. Kronenberg reported the use of immune gamma globulin in the treatment of uveitis. He said that the immunologic rationale and the favorable results obtained suggest the need for further investigation in this disease. The favorable results obtained in the treatment of retinitis pigmentosa with placental extract by Filatov was not corroborated by Dr. Kronenberg. Old corneal opacities, tuberculous episcleritis, diabetic retinopathy, and vascular changes

in the retina did not respond to treatment with placental extract.

Dr. Kronenberg also described the use of placental tissue coagulant, preoperatively, to lower the blood clotting time in diabetics.

Syndrome of glaucomato-cyclitic crises

Dr. Adolph Posner and Dr. Abraham Schlossman presented nine cases which form a homogenous group and exhibit signs of mild glaucoma, associated with cyclitis. The clinical features were as follows:

Unilateral attacks of ocular hypertension occur without congestion, pain, or marked impairment of vision. These attacks last from a few hours to two weeks. Cells in the aqueous and keratic precipitates appear either simultaneously with hypertension or within 24 hours after the onset of the hypertension. Posterior synechias are never observed. The precipitates are not numerous and disappear spontaneously within one month. Individual attacks may occur without precipitates. Heterochromia was present in one third of the cases and anisocoria in one third of the cases.

Gonioscopy showed the angle to be open in all four cases examined. In two of the cases it was wide, and moderately narrow in the other two cases. The attacks are self limited.

Pupillographic examinations gave normal curves in all six cases examined. In three cases the curve was of the type found in primary glaucoma, while the other three cases showed evidence of disturbances of the central sympathetic nervous system.

Dr. Posner said that treatment does not shorten the total duration of the attack. Pilocarpine (½ to 2 percent) usually lowers the tension. Homatropine is not dangerous and its action is not dependable. Surgery does not prevent recurrence of the attacks.

This syndrome of recurrent attacks of unilateral glaucoma with mild cyclitic signs forms a clinical entity intermediate between primary and secondary glaucoma, since it exhibits features of both types of the disease.

Discussion. Dr. Ludwig von Sallmann agreed with Dr. Posner and Dr. Schlossman that cases of patients with recurrent attacks of increased intraocular pressure as was described should be classified as a definite syndrome. He said that these cases can be distinguished from typical primary glaucoma with a few deposits on the cornea, from secondary glaucoma, and from the so-called sympathicus glaucoma with and without heterochromia.

In regard to the pupillographic examinations, Dr. von Sallmann raised the question as to whether, in view of the unilaterality of the lesion, a disorder in the subordinate ciliospinal center of Budge can be completely disregarded.

Dr. von Sallmann said that in addition to the hypothalamic disturbance, Dr. Posner assumes the coexistence of a peripherally operating mechanism as, for instance, a labile peripheral autonomic system; this factor must be present unilaterally. There seems to be a considerable range of doubt or alternate explanations. One could visualize that the functions of the terminal nerves or the capillariomotoric response of the other intraocular mechanisms of regulation are at fault in the one eye. In view of the presence of infective foci and strong allergic reactions of some of the patients, the question arises whether an antigen-antibody reaction or a toxic factor couldn't be responsible for the onset of the periodic attacks in a disposed eye. That is, the cells in the aqueous and the precipitates on the cornea could be the expression of a transient low-grade inflammation rather than of a primarily nervous disturbance.

What further studies may reveal on the interrelationship between disorders of sympathicus centers and between the centers and a peripheral mechanism remains to be seen. At the present time it is likely that a transient increase in the permeability of the

ciliary capillaries is the most important single peripheral factor, which has a bearing on the development of the recurrent attacks. In the last year, studies were conducted in the Knapp Laboratory on an experimental glaucoma induced by increased permeability of the ciliary capillaries. Various therapeutic approaches were studied on experimental animals. The effects of adrenergic drugs, of pituitrin, of substances acting on the intracellular cement, as Ca ions and adrenal cortical hormone, and of a series of histamine antagonists were tested, and promising results were obtained both in respect to the reduction of increased permeability of intraocular capillaries and the lowering of intraocular pressure. There is some hope that one or the other of these therapeutic measures will be of help in the glaucomato-cyclitic crises described by Dr. Posner and Dr. Schlossman.

Bernard Kronenberg,
Secretary.

SOCIEDAD OFTALMOLOGICA DE MADRID

May 16, 1947

Syndrome of Van der Hoove

Dr. Mier read a paper on Syndrome of Van der Hoove. The patient, 32 years of age and 1.38 meters tall, had suffered three fractures, which were corroborated by X-ray pictures. The X-ray films also showed considerable osseous rarefaction of the epiphyses of the long bones and a very small and dense sella turcica in the shape of a kettle and covered with a bony layer, in the interior of which was the strangulated hypophysis. The patient showed hypophyseal disturbances. Both the middle and internal ears showed otosclerosis.

Discussion. Dr. Marion Amat congratulated Dr. Mier on his brilliant dissertation and recalled the fact that he had published in the *Semana Medica Española* of December, 1940, an interesting clinical observation which presented the complete triad of the Syndrome of Van der Hoove; namely, blue sclerotic, multiple fractures and otosclerosis and, in addition, cranial asymmetry, alterations in the basal metabolism, and calcemia.

Visual disturbances as the first subjective manifestation of a tumor of the hypophysis

Dr. Marin Amat and Dr. Marin Enciso presented a paper on this subject.

Marfan's syndrome

Dr. Marin Enciso and Dr. Lopez Garcia presented a paper on "Marfan's Syndrome: Extraction of Subluxated Crystalline Lenses." They presented a girl who showed this syndrome with excessive length of the limbs, especially the hands, feet, and fingers, very large arms, congenital subluxation (ectopia) of both crystalline lenses complicated by ocular hypertension which necessitated an iridectomy in both eyes and later extraction of both subluxated, transparent crystalline lenses. The operations were accomplished without any loss of vitreous or any complication.

Discussion. Dr. Mario Esteban. Arachnodactylia is one of those dystrophies, the study of which is always interesting. The pathogenic interpretation of the condition lends itself to the formulation of hypotheses which leave behind them not one, but many questions.

Evidently there is an anomaly of development of germinal origin which affects the mesoderm. But what is the fundamental cause of the dystrophy? Why are there other malformations and ectopia of the crystalline lenses and such excessive length of the bones of the hands and feet that the flexor tendons become relatively short causing a permanent contraction of the finger joints?

One may suggest the theory that there is hypophyseal disfunction, and perhaps also

parathyroid disfunction, which start in fetal life.

Professor Carreras. Marfan's syndrome does not constitute an isolated symptomatic complex, but forms part of a much larger and more general syndrome for which Schmidt proposes the name of "Congenital Mesodermal Dysmorpho-dystrophy." Marfan's syndrome is one of its clinical types and another (at least until now) is the syndrome of Marchesani in which there is microphakia and spherophakia in addition to brachydactylia. There have been cases of brachydactylia with ectopia of the crystalline and of spherophakia with arachnodactylia, which proves there is some common basis for the aforementioned changes. At times there are frustrated and asymptomatic forms. In addition to luxation of the lens and spherophakia, which are the most characteristic features, there may exist other ocular anomalies (megalocornea, aniridia, coloboma, and so forth). Of the different theories proposed to explain this syndrome, the mesodermal theory of Weve seems best.

The disturbance which affects the development of the mesodermal structures could be explained by the action of some chemical or prehormonal substance, since in the embryonal period, when these events seem to begin, one cannot speak of actual hormones. Delayed involution or partial persistence of the tunica vasculosa lentis could originate these zonular deficiencies which would later cause the changes in the crystalline lens. Although the lesion attacks ectodermal organs and formations, such as the crystalline, the origin of the disturbance is rooted in the mesoderm. Heredity also plays a role. The typical cases of the disease, or even the incomplete and frustrated forms, are transmitted by heredity of the dominant type. The asymptomatic forms, on the other hand, are generally transmitted by the recessive type.

Dr. Marin Enciso (closing). In the first place I wish to thank Dr. Esteban and Professor Carreras for their remarks on the subject. With reference to Dr. Esteban's suggestion on the endocrine origin of the syndrome, this has been partly answered by Professor Carreras. The disturbance begins in embryonic life at a time when one could not very well speak of fetal endocrine influences. The morphologic peculiarities of the patients with Marfan's syndrome suggest, however, the influence of endocrine factors, especially of the hypophysis, but actually this idea can be only a suggestion, since it is not based on definite data.

With regard to the substance which is affected, it seems that in Marfan's syndrome the changes in the mesenchyme dominate to such an extent that some include it in the congenital mesenchymal dysplasia. However, it is not rare to observe cutaneous and neurologic changes which lead one to think of simultaneous changes in several blastodermal parts. The disturbance probably depends on a juncture of genetic factors combined in diverse ways in various cases.

Joseph I. Pascal,
Translator.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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TERRY-OWENS DISEASE

Since retrolental fibroplasia was described by Terry in 1942, our knowledge of this disease of premature infants has been fragmentary, and conflicting hypotheses have been advanced in an attempt to explain it. These hypotheses, have been based upon (a) clinical observations of patients seen in advanced stages of the disease, and (b) upon histologic examinations of eyes removed when the lesions were far advanced. It is natural, therefore, that these examinations made during the late stages of the disease have led to inadequate and conflicting premises as to its nature.

To understand retrolental fibroplasia, it is necessary to follow the clinical course of the disease from its onset to the development of the final characteristic picture. Dr. William C. Owens and Dr. Ella U. Owens have investigated the problem of retrolental fibroplasia from this standpoint. Their findings will be reported in an article entitled, "Retrolental Fibroplasia in Premature Infants," which will appear in a subsequent issue of the JOURNAL. As part of their study

on retrolental fibroplasia, they routinely examined all the premature infants weighing 2,000 grams or less born in The Johns Hopkins Hospital or admitted to the Premature Nursery of The Johns Hopkins Hospital for a period of two years.

In their report they describe the clinical course of retrolental fibroplasia as it occurred in 10 premature infants in whom they observed the lesion develop. In every case observed, the lesion had its onset after birth. The lesion did not result from an overgrowth or persistence of any structure characteristic of an embryonic or fetal stage of development such as the tunica vasculosa lentis or the primary vitreous, as had formerly been supposed. They found that the earliest changes occurred in the retina and the retinal vessels. The retinal vessels became enlarged and tortuous; the retina became gray, swollen, and elevated; and the vitreous became hazy. As the disease progressed, large masses of elevated retina fused and gradually covered the retrolental space, producing the picture that has been described as typical of retrolental fibroplasia, which in reality is the end stage of the lesion. In the cases described, the lesion began to develop about two months after birth. It gradually increased in activity and intensity until about the fifth month after birth, when it began to subside, the later changes being due to contracture of the retrolental tissue.

The article of Dr. William C. Owens and Dr. Ella U. Owens is particularly interesting for their observations throw an entirely new light on the nature of retrolental fibroplasia. The disease has a definite onset in postnatal life. It occurs primarily in the retina and retinal vessels; it is not related to an abnormal persistence or hyperplasia of the tunica vasculosa lentis or of the primary vitreous. The fact that the lesions in some of the cases subside spontaneously suggests the possibility that, if the cause of the disease can be found, the institution of proper therapy may result in a regression of the lesions, leaving eyes with almost no remaining abnormality.

The disease that Terry described and upon which Owens has elaborated so convincingly is, therefore, surely an entirely separate entity from that of the overgrowth or persistence of fetal developmental tissue within the eye. In discussing the paper by Owens at the recent meeting of the Wilmer Residents Association, Verhoeff pointed out that the term "retrolental fibroplasia" is not truly significant of either of these two conditions, and thus its employment adds to the confusion. Although eponyms are unscientific, the use of the term "Terry-Owens Disease" can be practiced in this event until a more suitable, or at least scientific, name is evolved.

Much pride is taken in proposing that the name "Terry-Owens Disease" be made an integral part of our medical and ophthalmic nomenclature. Tularemia conjunctivitis was the first ophthalmic disease discovered by ophthalmologists in the United States and "Terry-Owens" disease is the second. We are growing up.

Derrick Vail.

COLOR BLINDNESS; NO CURE

During the examinations in the enlistment and drafting of men and women for admission to the armed forces—Army, Air, and especially the Navy—many of them were astonished, not to say shocked, when informed that they were "color blind." Because they had never experienced difficulty in their lives, and, doubting the recruiting examiner's judgment, they sought examination elsewhere. When convinced that they had shown uncertainties in their estimates of the colors shown them, they were inclined to believe they were ignorant of colors, or that they had some temporary and unsuspected diminution of their sight in general. They sought means for educating themselves in the appreciation of colors, adopt-

ing exercises by which they might so correct their defects as to be able to pass the prescribed tests.

The term "color blindness" is a rather cruel designation of the state of the sight which prevents an individual from easily recognizing colors and makes him hesitate to declare the name of a color and to display an ability to match it and blend it with other colors.

The appreciation of *color* is a physical property of the sight—occasioned by the breaking up of light when broken into rays of differing quantities. This action is shown when one holds a prism of glass before the light as it comes from the sun and observes the reflection thrown into various colors, like those of the rainbow after a storm, exhibiting the red at one end and violet at the other. Now, there are some persons who cannot see red, or green, or violet; these are the true actual so-called color blind. And there are others who have difficulties in appreciating some colors when they are shown under varying conditions of light, or in matching or blending a given color with another. Few, very few, persons are absolutely blind to all the colors.

Scientists have given another name to color blindness—it is spoken of as "achromatopsia," and those who, while recognizing all the colors as such, have difficulty in manipulating any or all of the rainbow colors are the "anomalous." These anomalous persons have been divided into classes depending upon the number of the colors with which they have difficulty, or the replies which they make in the testing—thus "deuteranopes" and so on in each degree, the main color is given a Greek numeral to indicate the result. There is no blindness in the ordinary sense.

In practice and experience in railway, naval, and other forces, red and green have been the colors employed as signals of safety or danger. So, in general, because of the employment of those two colors, such achromatics have been spoken of as the "red-green blind." This defect has long been recognized and largely has had a place made for it, since the subjects, as a rule, exhibit the defect in all their avocations. Those who, in bright light make out to the satisfaction of all, are prone to go astray when in reduced light, or in cloudy weather, or at sundown and daybreak. This class is more dangerous in action than the known red-green individuals as they cannot instantly distinguish the colors set.

The cause of color blindness is not known; many theories are held, and it is amusing to stand on the sidelines and watch the fierceness of the fights which go on between the advocates of one adopted theory who struggle to have opponents accept their pet ideas. As a working hypothesis, most ophthalmologists are willing to accept the Young-Helmholtz theory that each color has its own nerve, the rays carried over it to be appreciated by the brain.

At present, ordinary cases of color blindness present no visibly known cause for their trouble. Their eyes are healthy and show no departure in structure from what is obtainable in the examination of the perfectly normal cases. But, color deficiencies are complained of by those who have had distinct disease of their eyes, or of the nervous system, as in optic atrophy and in some cases of cataract.

The appreciation of color was recognized in the earliest known records of humanity. The recognition of the "Arch in the Heavens" was an index of man's ability to distinguish the different bands in the rainbow. At the time of the Flood, doubtless, not all the colors as known to man today were recognized by primitive man. Even nowadays savage peoples in the remote lands do not employ all the civilized colors. We may conclude that *color* has been a developed sense. May we hope that, in the future, the civilization of the, until recently, savage inhabitants of the Oceanic Islands can be observed by

scientific inquirers so that many new facts concerning the color vision of such backward peoples can be made known to us?

It was a common expression of those who had been rejected by the Armed Services, that they were "color-ignorant." It is true that some persons do not know what names to give to modern tints and shades of common colors, they cannot say what is "cerise" or what "mauve"; yet, these same individuals, when shown the red, green, blue, yellow, and white lanterns in the railways and street crossings, accurately and promptly called the names of the colors.

Now, because of having been rejected, many, eager for enlistment or employment, have sought means to educate themselves or so to develop their senses that they can pass the same tests which they failed to pass a short while before.

Color blindness has been closely observed since about the end of the 18th century. Many are the ludicrous stories told of well-known persons' mistakes, such as a staid Quaker appearing at First Day's breakfast in a flaming red coat of his own selection, having indignantly objected to the practice of his wife's insistence on choosing the cloth for his clothing. Or, the more serious story of a trusted railway engineer, who for 40-odd years had guided his train to a distant city without a broken record. He declared that he "knew every tie from the one point to the other with his eyes shut," but when examined after a short illness, was found to be deficient "with his eyes open."

Color blindness is found in males, rarely in females, but a woman of a color-blind family may be a "carrier" and transmit the affection to her male descendants, the females of the family serving as carriers to their male children.

At the beginning of the scientific study of color vision, common colored articles were employed. Since these did not have fixed qualities, efforts were made to standardize the coloring of objects employed and to have the standards accepted by all interested nations as the international standard.

Lamp lights were devised and wool skeins dyed to a fixed degree. Later cards were printed. All these were first standardized and then modifications of them were adopted, and "mistaken colors" arranged so as to enable the examiner to have an idea of what the defective one might offer as his conception of the true colors. The cards, or plates, have various figures formed of the test-color, surrounded by similar color formations but of the "confusion color." This makes the set figure indefinite and has the property of confusing the examinee, thus demonstrating to the examiner the true state of his vision.

To repeat, it has been the endeavor of the true scientist to seek means at least to increase a deficient's powers, but without success. It is to be recorded, alas, that designing persons, or perhaps misguided enthusiasts, have prepared schemes arranged with standard and obscuring colors, with which they have held out to would-be "clients" assured prospects that by daily practice with these devices, they can "cure" this color blindness and safely pass all the color tests prescribed by the Government.

I have been concerned all my medical life with practical observation of known and suspected color deficiencies—my professional observation confirming facts I had become aware of in my early days and in school and college life. In deep sympathy for those found to be so defective that they have been forced to leave their chosen, and otherwise efficient, employment because of their "color blindness," I have pursued every available means to be practiced for such studies. My experience has been in practical-every-day environment, not in complex laboratory surroundings. In no case have I been rewarded by results so perfect or of such cheering success that a color-blind man or a woman, under all sorts of conditions devised, has been

able to pass the tests conducted by another disinterested observer. In the cases of excessive use of tobacco, especially of an alcoholic, there have been recorded recoveries from that kind of toxic amblyopia after a season of abstention from "nicotine and its tincture."

In May, 1947, there was published in the *Archives of Ophthalmology* an extensive report by Roswell Gallagher and his colleagues of their analyses of the "Efforts of Training Methods on Color Vision" observed in the case of 40-odd individuals in which they state:

"Training which enables a person (previously classified as a deficient in color vision) to achieve a test rating comparable to that of persons who have normal color vision apparently does not result in the development of the ability to differentiate colors in all situations and under all conditions with the accuracy and speed possessed by those who have normal color vision." When testing objects, not hitherto employed, were exhibited, the subject failed to recognize them correctly.

And, at the meeting of the American Ophthalmological Society held in June, 1947, at Hot Springs, Virginia, it was unanimously ruled that—"The conclusion reached after consultation with various groups interested in the subject of defective color sense was 'to the effect that color deficiency cannot be remedied by diet, training, or any other treatment now known to Science.'" This judgment was confirmed by the American Medical Association, and was repeated at the meeting of the association at Atlantic City in June, 1947.

It is by reason of these pronouncements that this paper has been offered for the benefit of those who might have been induced to enter into schemes devised to improve their color defects.

Burton Chance.

OBITUARIES

ERNEST F. KRUG
(1877-1948)

Dr. Ernest Frederick Krug died on February 28, 1948, after a long illness. He was born in Cleveland, Ohio, a son of Prof. Joseph Krug, supervisor of instruction in the German language in the Cleveland public schools, and of Magdalena Weinhardt Krug.

After completing his education in Cleveland, Dr. Krug came to New York to study medicine and was graduated from the College of Physicians and Surgeons of Columbia University in 1900. He then interned at the Lennox Hill Hospital in New York and from that time on was always associated with that institution.

He became an assistant to Dr. Emil Gruening and later to Dr. Wilbur Marple. He also took postgraduate work abroad, studying for some time under the eminent ophthalmologist Prof. Dr. Ernst Fuchs. He successfully practiced ophthalmology and otolaryngology for many years, but from 1922 on he limited his practice to diseases of the eye. During World War I, Dr. Krug served in the medical corps. He held the rank of major.

After fulfilling various appointments at the Lennox Hill Hospital, Dr. Krug became attending ophthalmologist in 1920, from which position he was retired, in 1942, to become a consultant in his department. Through his untiring efforts, a modern and well-equipped eye department was established and he devoted a great deal of his time to teaching and training the younger ophthalmologists. He was always interested in the younger men and was happy to pass on to them the knowledge which he had gained from his long years of experience. He was always willing to offer them guidance and encouragement. Many of these doctors continued throughout his lifetime to seek his advice and often called him in consultation.

Dr. Krug held the appointment of associate professor of ophthalmology at the Post Graduate Medical School for many years and, until 1946, he was associate attending ophthalmologist at the Post Graduate Hospital and Dispensary. He also served for some time as attending ophthalmologist at the Central Neurological Hospital at Welfare Island.

He was a Fellow of the New York Academy of Medicine, a member of the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology, and the New York Ophthalmological Society.

Dr. Krug was a thorough ophthalmologist in every field, a keen observer, an accurate diagnostician, and a very skillful surgeon. His methods were simple and direct. He loved his chosen work deeply and he gave unstintingly throughout his long career of himself and of his knowledge to those who were afflicted. He will be remembered by all who knew him for his genial good nature, ready wit, and quick repartee.

His contributions to ophthalmic literature were few, but they were important. They were as follows: "An investigation of the ferment activity of the rabbit's vitreous" (1931); "Foreign substances injected into the vitreous of the rabbit," Ernest F. Krug, M.D., and George L. Rohdenburg, M.D. (1932); "Venous angioma of the retina, optic nerve, chiasm and brain," Ernest F. Krug, M.D., and Bernard Samuels, M.D. (1932); "A cyst of the posterior chamber with a microscopic study of the eye" (1937); "A case of tuberous sclerosis," Ernest F. Krug, M.D., and Francis A. Echlin, M.D. (1943).

In 1911 Dr. Krug was married to Miss Louise P. Hoffmann, who survives him. He also leaves two sons and three grandchildren.

Joseph H. Krug.

WILLIAM TARUN
(1870-1948)

Dr. William Tarun was graduated from the University of Maryland Medical College in the class of 1900. He began his special training at the Presbyterian Eye, Ear, and Throat Hospital almost at once under Dr. J. Frank Crouch in otology and later under Dr. Hiram Woods in ophthalmology. Dr. Woods shortly appointed Dr. Tarun chief of the Eye Clinic at the University Hospital and associate professor of ophthalmology and otology which position he held until Dr. Woods retired from the university.

In his earlier years of practice, Dr. Tarun had a large following in both otology and ophthalmology but later limited his practice to ophthalmology. With his keen mind and excellent training he soon became one of the leading ophthalmologists of Baltimore.

He was a member of the American Medical Association, the American College of Surgeons, the Medical and Chirurgical Faculty of the State of Maryland, and the American Ophthalmological Society to which he was elected in 1917 and whose meetings he always attended.

His original contributions were not numerous but he was active in the discussion of the papers presented. His hobbies were golf and gardening in both of which he excelled.

Dr. Tarun was an able physician, a good citizen, a Christian gentleman, and a most genial companion. His relationships to his colleagues were always characterized by the highest ethical standards. His passing followed a distinguished career of work well done, and his memory will long live in the hearts of his many friends and patients.

Clyde A. Clapp.

ERVIN TÖRÖK
(1877-1947)

By the death of Ervin Török, ophthalmology has lost an accomplished surgeon

and a man of attractive personality. Dr. Török was a native of Hungary, where he was graduated from the University of Budapest and received his doctor's degree in 1899. He obtained his preliminary education in ophthalmology at the University Clinic of Schulek in Budapest. After several years of postgraduate training with Grosz and Blaskovics in Budapest and with Hirschberg in Berlin, he arrived in the United States, in 1905, with a letter of introduction to Dr. Herman Knapp. He then became resident and a member of the staff at the New York Ophthalmic and Aural Institute where he inaugurated a tuberculin clinic and was one of the first to use this method of treatment in this country. He was a successful teacher in the postgraduate course of instruction which was then being given at that institution. He became full surgeon in 1913, when the hospital moved to 57th Street and its name was changed to the Herman Knapp Memorial Eye Hospital. He resigned as surgeon, in 1927, to become professor of ophthalmology at the New York Polyclinic Hospital.

In 1912, Dr. Török was appointed ophthalmologist at the Beth Israel Hospital in New York City; he organized the department of ophthalmology at this rapidly growing hospital and remained its chief for 25 years, until his resignation in 1938. It was here that he showed his ability as an organizer and capable administrator.

His other hospital affiliations included the Beekman Street Hospital and several institutions in Westchester County. His excellent clinical knowledge associated with an unusually clear and logical approach to ophthalmic problems won for him a very active field as a consultant for several of the larger accident insurance companies and as expert witness in medico-legal claims.

He became a member of the American Ophthalmological Society in 1912.

Dr. Török published some excellent papers, mostly on surgical subjects, and he was the author, together with Dr. Gerald H. Grout, of "Surgery of the Eye: A Handbook for Students and Practitioners," which was published in English in 1913; a second edition appeared in 1925, and was translated into Spanish. The book describes the surgical technique of the Blaskovics clinic.

A sound and resourceful surgeon, his skill in operating was striking. He was one of the first in this country to perform the scleral resection (Müller's operation) for retinal detachment in high myopia. He also modified the method of forceps intracapsular extraction by grasping the anterior capsule of the lens with a blunt forceps and then extracting the lens by traction and by exerting pressure according to the usual von Graefe procedure.

He was a man of attractive presence and of an exceptionally charming personality. Always courteous, never ruffled or impatient, his tender sympathy endeared him to his patients by whom, and by his many colleagues, his passing will be greatly deplored. He is survived by his wife, who was Louise B. Felter.

Arnold Knapp.

CORRESPONDENCE

Dr. van Heuven replies

Editor,
American Journal of Ophthalmology:

In reply to the correspondence on page 484 of volume 31, April, 1948, American Journal of Ophthalmology, I want to state that I take no responsibility for the report of my lecture on "Retinal Detachment," held at Cleveland's Ophthalmological Club in January, 1947. Further:

That I was dismissed from lecturing at the Utrecht University by the Germans on Monday, April 12, 1943, and, after the liberation of Holland, was restored, in 1945, by Royal Appointment;

That I never secretly subscribed to the Nazi organization of physicians;

That indeed two scientific papers from my hand appeared in German scientific journals; the copy of these, however, had left my house before the outbreak of the war;

That together with all patriotic colleagues I resigned my membership of the Dutch Medical Association during the war, and that after the liberation I have never applied for membership again on account of disagreement with their leaders (I shared this view with several distinguished colleagues);

That I still am a member of various scientific Dutch societies. I enclose translation of a statement of the secretary of the Dutch Ophthalmological Society that my resignation of membership had nothing to do with politics or political dispositions.

During the war I was a regular member of the Dutch Medical Underground sharing all their activities and being penalized for it by the Germans. Besides I played an active roll in the Fighting Underground. I enclose a statement from my direct superior as to these, my activities.

I regret that a young oculist, whom I only met once or twice and who has no personal knowledge of my affairs, has badly confused gossip and gospel and made himself the speaking trumpet of others.

(Signed) J. Alexander van Heuven,
New Haven, Connecticut.

STATEMENT FROM DR. ROELOFS

I, the undersigned, Dr. C. Otto Roelofs, secretary of the Dutch Ophthalmological Society, declare herewith that the reason why Dr. J. A. van Heuven, ophthalmologist at Utrecht, decided to resign his membership of this Society in October, 1945, had *nothing* to do with politics or political dispositions.

(Signed) C. Otto Roelofs,
Amsterdam, Holland.

STATEMENT FROM UNDERGROUND SUPERIOR

During the occupation there was in my house a center for the food supply and armament of the underground forces. For many years I knew Dr. van Heuven as a patient and when I approached him with the question whether he would dare to do something on behalf of the underground work he did not hesitate for a moment and at once consented.

We all trusted him fully; right from the beginning he was in perfect knowledge of everything which happened at my house. He took an active part in the food supply of the underground movement and had to do many jobs which were most unfamiliar to him, paid our secret caterers, and was present at our daily conferences.

When the commander of the first Division, Leo Berk, was shot and we were unable to find him, Dr. van Heuven searched all the hospitals as a doctor to find Berk and did not even hesitate to penetrate for that purpose into the St. Anthony Hospital which was entirely occupied by the Germans. In identifying men who were murdered by the Huns he has rendered great services to us.

We were very painfully struck by the fact that, after the liberation, various gossip was told about Dr. van Heuven, while we knew for certain that this man was politically perfectly trustworthy.

I am always willing to provide any further explanation wanted.

(Signed) W. J. M. Bordewijk,
Utrecht, Holland.

PREDILECTION OF LEFT EYES FOR GLAUCOMA

Editor,
American Journal of Ophthalmology:

In the October, 1947, number of the JOURNAL, Holst published a statistical study of glaucoma in which he shows the incidence of glaucoma to be higher in the left eye. Ascher, one of the leading experts of

ocular hemodynamics, explains for this fact in the Correspondence column of the February, 1948, issue of the JOURNAL.

I think that it is only possible to decide a question of such importance on the basis of a large number of cases. In 1935 I published in *Klinische Monatsblätter für Augenheilkunde* the most extensive statistics of glaucoma based on 3,214 cases of the Budapest University Ophthalmic Clinic. In these statistics the incidence of occurrence in the right or in the left eye was not taken into account. Holst's publication inspired my revision of the 1935 publication completing it with the material from the Eye Department of the Jewish Community Hospital in Budapest.

I have found monocular glaucoma in 669 cases. Bilateral glaucoma in which that of one eye was absolute occurred in 761 cases. Of the monocular cases, 359 occurred in the right eye and 310 in the left. Of the bilateral cases in which absolute glaucoma was present in one eye, 363 occurred in the right eye and 398 in the left.

Consequently it cannot be stated that, as regarding glaucoma, the left eye would be in a less advantageous position.

(Signed) P. Weinstein,
Budapest, Hungary.

BOOK REVIEWS

NEUROLOGY OF THE OCULAR MUSCLES. By David G. Cogan, M.D. Springfield, Illinois, Charles C Thomas, 1948. Clothbound, 229 pages, 121 illustrations. Price, $6.00.

In his preface, Dr. Cogan finds himself ". . . in the ambiguous position of simplifying in the interest of lucidity and yet being critical of simplification in such a complex field as the ocular motor system." He has succeeded extremely well in attaining his goal and has done it in such a pleasing style that one fails to hear the wheels turn.

The chief disappointment, if there be one, is in the brevity of the book. Upon finishing it, the reader is left with the definite impression that Dr. Cogan had much more to say, but hesitated to do so for fear of making statements which might later prove to be erroneous. The desire of the student is always for a pedagogic presentation and there is comparatively little pedagogy in the book.

The practicing ophthalmologist will find much of interest and the neurologist even more. The first section of the book is concerned with the anatomy and physiology of the ocular muscles. The next section is on the anatomy of the cranial nerve supply. A third section is devoted to supranuclear connections of the ocular motor system. The last two sections deal with the pupil and nystagmus respectively.

Two points make the book extremely worthwhile to all who are even remotely interested in the neurology of the ocular muscles. These are: first, the superb illustrations and, second, the very comprehensive bibliography containing 825 references in many languages. The only fault, in the mind of the reviewer, is a very small one. It is in the use of the term "ductions" rather than the more preferred one of "vergences" —at least if one follows the ideal of Lancaster.

Every ophthalmologist is sure to find the book a valuable addition to his library and one which will doubtless whet his appetite for more knowledge along the same lines. We can only hope that the second edition will be twice as thick as the first.

Richard G. Scobee.

TRANSACTIONS OF THE SOCIÉTÉ BELGE D'OPHTHALMOLOGIE. Meeting of December 1, 1946, pp. 1-174.

This volume of the Transactions of the Société Belge d'Ophthalmologie brings the membership lists of the society, the minutes of the business meeting, the reports of the scientific session, and the obituaries of Dr.

H. Coppez, Dr. L. Leplat, and Dr. J. Moorkens. At the scientific meeting 20 papers and case histories were presented.

L. Weekers and R. Weekers discussed nonperforating cyclodiathermy. M. and J. Wibo combined iridectomy with a preceding coagulating cyclodiathermy, using the pyrometric electrode. They reported on 10 patients in whom the operation was a success.

P. Huwart reported his striking success with sulfosine (sulfur in oil) in otherwise refractory cases of ocular syphilis. F. Rousel presented his studies on the intraocular application of penicillin in discussing his animal experiments and three clinical cases. He recommends the injection of penicillin into the anterior chamber in severe intraocular infection. Injections into the vitreous are extremely dangerous and should be used only as a last resource in cases of endophthalmitis.

R. Rubrecht's surgical treatment of active corneal lesions by partial excision of the diseased corneal tissue and superficial conjunctival flaps met with some opposition as in previous publications.

Among rare case histories was one by Elise Soil who removed a door handle from the bottom of an orbit without injury to the eyeball. De Walsche presented a case of an extremely large corneal papilloepithelioma. F. Bonhomme described the present technique in the fitting of contact lenses and indications for prescribing them. L. Alearts found syphilitic reagins in the anterior chamber and outlined their diagnostic possibilities in this preliminary report. He also presented a patient with larvae on Descemet's membrane.

Other presentations were made by M. Appelmans and N. Lanoy on superficial punctate keratitis in onchocerciasis and by Jean Coppez on nummular keratitis.

Alice R. Deutsch.

TRANSACTIONS OF THE OPHTHALMOLOGICAL SOCIETY AT PARIS AND OF THE OPHTHALMOLOGICAL SOCIETIES OF THE EAST, OF LYON, AND OF THE WEST. Meetings of March-April, 1947. No. 2, pp. 120-294.

This issue brings a résumé of the meetings of the Ophthalmological Society at Paris on March 15, 1947, and April 19, 1947, and of the Ophthalmological Society of the East on March 16, 1947. It is divided into three parts. Each part gives a report of the scientific sessions of one day.

The first part contains 10 papers about various topics. Three papers deal with the experiences with penicillin. Dubois-Poulsen and Vouters describe its effect in metastatic uveitis; Guilloumot and J. P. Baillart have used this drug in orbital osteoperiostitis; Carlet-Soulages summarizes his experiences with penicillin on gonococcus conjunctivitis when he was stationed in the Far East with the French expeditionary forces.

A. Dubois-Poulsen's and A. Rozan's graphic study with Howe's ergograph on the ciliary muscle and the relationship of the objective signs of fatigue of this muscle to asthenopic complaints are very interesting. So are M. R. Onfray's presentation of Dvorine's color plates and Peron's and Desvigne's case report of a periarteritis nodosa, localized on the arteries of the conjunctiva.

The second part contains the minutes of the business meeting and 10 papers of varying importance. P. Halbron, F. Lepage, Leconte, and H. Mowas report on the substitution of penicillin for silver nitrate in the prophylactic treatment of the newborn, an especially interesting topic because of similar research work in this country. E. Haas discusses the characteristics of gonococcic iridocyclitis, its specific reactions and treatment. P. Michaud emphasizes the importance of mountain air and rest in ocular tuberculosis. There were also case reports on

chorioretinitis and periphlebitis in hereditary syphilis; of uveitis in leptospirosis; and of a choroidal angioma complicated by intractable glaucoma which followed an injection of tetanus antitoxin.

A symposium on the essential shrinkage of the conjunctiva was the highlight of the session on March 16, 1947. Beauvieux delivered the lecture which dealt with the clinical description, pathology, etiology, differential diagnosis, and treatment of this comparatively rare disease.

The topics of the following 17 papers were many and their presentation and subjects of varying interest. A. Bronner and P. Pouliquen gave another lecture on penicillin therapy with especial emphasis on the technique of subconjunctival, anterior chamber, and vitreous injections in otherwise hopeless external eye infections.

Mathieu describes an original modification of intracapsular cataract extraction of an intumescent lens wherein a micropuncture to decrease the tautness of the capsule precedes the extraction. R. Bruckner combines a case report on toxoplasmosis with a detailed study of this disease. The origin and the manifestation of uveal carcinoma were discussed by J. Nordmann and G. Hoerner, and sarcoma of the choroid by J. C. Poirot.

A detailed case report of a nevocarcinoma of the lacrimal sac was given by Gallois and Michel-Gallois. Two cases of perforating scleromalacia were described by C. Henry and B. Algan. Beau, Thomas, and Bénichaux report a case of bilateral pulsating exophthalmus and explain the pathology and the treatment, which consisted in the ligature of the right common carotid. The pathogenesis of the ocular structures in methyl-alcohol intoxication was the topic of Rohr's paper.

The closing paper was read by Christian Henry. He reported on his research concerning the diastolic hypotension of the central retinal artery in fatal coma. He stresses the importance of the diastolic retinal pressure on the outcome of every kind of coma. A comparatively high diastolic pressure is of favorable prognosis as it is the sign of a cellular edema which is reversible. A decreasing diastolic pressure indicates cerebral degeneration which continues unrelentingly toward death. Even so the toxic cause should be removed. Alice R. Deutsch.

LA PROPHYLAXIE DE LA CÉCITÉ EN ROUMANIE (1940-1947). By Dr. Nicolas Blatt. Bucureşti, Imprimeria Naţională, 1947.

In this 68-page monograph, Blatt discusses the problem of blindness in Roumania. He briefly displays statistics, considers the causes of blindness, and gives a critical review of such social measures as the laws that apply to vaccination against smallpox, the application of the Credé procedure, and industrial accidents, schools for amblyopeš, pensions for the blind, eugenics, and social insurance.

F. H. Haessler.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Crosby, E. C., and Henderson, J. W. **The mammalian mid-brain and isthmus regions. Part II. Fiber connections of the superior colliculus. B. pathways concerned in automatic eye movements.** J. Comp. Neurol. **88**:53-92, Feb., 1948.

The paper deals primarily with the pathways underlying certain automatic eye movements. The studies were made on the midbrain of the monkey. The authors stimulated selected areas of the exposed brain by a minimal faradic current and noted the eye movements produced.

Francis M. Crage.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Ajo, Aarni. **On the refractive index of the retina.** Acta Physiol. Scandinav. **13**: 130-149, 1947.

The author's measurements were made on the retinas of cows, newly born calves, pigs, and horses. The refractive indices for these animals are reported. The values were higher in the light adapted retinas since the visual purple exudes water when bleached.

Francis M. Crage.

Barany, E. H. **The action of atropine, homatropine, eserine, and prostigmine on the osmotic pressure of the aqueous humour.** Acta Physiol. Scandinav. **13**:95-102, 1947.

Aqueous solutions of these drugs were instilled into the conjunctival sacs of male albino rabbits. Atropine and homatropine showed a tendency to lower the osmotic pressure of the aqueous. Eserine and prostigmine caused a reduction in the osmotic pressure in previously untreated eyes. An initial phase of rise in intraocular pressure and intense iris hyperemia was followed by a hypotonic phase in which the tension was lowered and there was increased protein content and visible flare in the aqueous. The protein increase and flare were attributed to the increase of capillary permeability. In the previously treated eyes these miotic drugs produced none of these changes except a moderate reduction in the intraocular pressure. In these eyes, habituated to eserine and prostigmine, the osmotic pressure remained practically unchanged. Increased capillary permeability

was considered as the probable cause for the decrease in the osmotic pressure.

Francis M. Crage.

Babudieri, B., and Grignolo, A. **Three new methods of micro-dosage of penicillin.** Reports of the Superior Institute of Health. V. 10, 1947.

The authors describe three new and simple methods for the biological micro-dosage of penicillin. These methods make it possible to assay the penicillin contained in a volume of 0.05 to 0.002 cm.^{3} and are so sensitive as to detect as low an amount as 0.000004 O.U. of the antibiotic, equal to only 2×10^{-12} gr. of pure sodium salt of penicillin.

Eugene M. Blake.

Bachi, S. and Borsello, G. **The effect of olfactory stimulation upon retinal arterial pressure.** Rassegna ital. d'ottal. **16**:418-432, Nov.-Dec., 1947.

The author studied the retinal arterial pressure following the use of various types of odorous stimulants and found that purely olfactory stimulants do not affect the pressure at all, while those substances which act upon the trigeminal nerve elevated the retinal pressure in a high percentage of cases. No effect was observed in anosmic individuals. This would indicate that there is no synergistic action between the olfactory and the trigeminal nerves as a result of olfactory stimulation by odors. Eugene M. Blake.

Friedenwald, J. S., and Woods, A. C. **Introduction and outline to the studies on the physiology, biochemistry, and cytopathology of the cornea in relation to injury by mustard gas and allied toxic agents.** By members of the staff of the Wilmer Institute. Bull. Johns Hopkins Hosp. **82**:81-101, Feb., 1948.

The preliminary studies showed that intracorneal injection caused more severe damage than did dropping the toxins on the cornea. Some products cause damage only when used in massive concentrations and others will cause damage in small doses. The recognizable clinical and histologic changes which follow chemical damage of the tissue are only remotely and indirectly connected with the initial chemical injury. Among many facts discovered; the excellent tolerance of the ocular tissues for di-isopropyl-fluorophosphate is worthy of special note. In mustard injury to the cornea it was felt that antibacterial agents could be given without danger of increasing the injury and would protect the eye from secondary infections. An effective ointment base in which BAL could be incorporated was found. Mustard was found to penetrate the cornea more rapidly than it did the skin. All compounds which were found most effective as antidotes to mustard contain sulfhydryl groups whose acid dissociation is below pH 7. Threshold doses of mustard cause transitory inhibition of mitotic activity in corneal epithelium. Glutathione in the intact cell is relatively inaccessible to mustard. The inhibition of mitosis is the most sensitive index of mustard injury. Studies on cohesion of epithelium with the stroma were undertaken because a loosening of the corneal epithelium is one of the earliest pathologic findings. Studies on corneal metabolism show that mustard causes a diminished oxygen consumption. There is a close metabolic interaction between the epithelium and stroma especially in the utilization of glucose and lactate. (1 table.)

H. C. Weinberg.

Friedenwald, J. S. **Summary and some possible interpretations.** Bull. Johns Hopkins Hosp. **82**:326-337, Feb., 1948.

In this paper the effects of mustard on corneal metabolism and the possible rela-

tion of these effects to the loosening of the corneal epithelium are discussed.

The metabolic interaction of corneal epithelium and stroma is reviewed, and it is emphasized that the epithelium normally assists the stroma in maintaining a supply of carriers for energy-rich phosphate transfers. There appears to be a special hydrogen transport system for the utilization of the stroma lactate independent of that concerned with the utilization of endogenous lactate in the epithelium. Pyruvate transfer and utilization remains unaffected after mustard injury to the cornea. Lactate and serine transfer and utilization is disturbed after mustard injury. The metabolic factors involved in lactate utilization are discussed.

The cohesive system is highly complex and there is a correlation between the loosening of the epithelium after mustard injury and the utilization of lactate from the stroma. Mustard causes loosening of the tissue under aerobic conditions but not under anaerobic. The boundary of epithelium and stroma may contain components of the hydrogen transport system for the utilization of stroma lactate. The oxidation of some of these components destroys some of the cohesive forces. Mustard injury appears to affect the nuclear boundary early.

H. C. Weinberg.

Friedenwald, J. S. **Note on karyolysis of the corneal stroma cells.** Bull. Johns Hopkins Hosp. **82**:178-181, Feb., 1948.

A method is described for preparing the cornea for the study of its stroma.

Extra fluid must enter the stroma to enable the nuclei to swell. This is obtained when the epithelial and endothelial barriers are injured. After exposure to mustard and other injurious agents the nuclear boundary becomes more fragile. The nuclei burst after exposure if incubated ten hours at 37 degrees C. Large doses of mustard or nitrogen mustard cause pycnosis of all types of corneal cells. (4 figures.) H. C. Weinberg.

Friedenwald, J. S., and Buschke, W. **Nuclear fragmentation produced by mustard and nitrogen mustards in the corneal epithelium.** Bull. Johns Hopkins Hosp. **82**:161-177, Feb., 1948.

Mustards and nitrogen mustards applied to the cornea cause a small percentage of cells in the basal layer to die by nuclear fragmentation. Pathologic mitosis occurs which is similar to the prophase and metaphase of normal mitosis. Lowered temperatures and anoxia cause inhibition of normal mitosis and inhibition of nuclear fragmentation of corneal epithelium exposed to mustard. Immersion of the tissue in Ringer's solution suppresses nuclear fragmentation and mitosis. Cells injured by mustard in the premitosis stage go on to nuclear fragmentation which is a pathologic mitosis. Because of injury the cells can not go back to the resting state. Exposure to hypotonic solution, solutions of low pH, and to nitrogen mustard dissolved in hypotonic solutions of low pH, causes an increase in the number of cells susceptible to this injury of nuclear fragmentation. (2 figures.) H. C. Weinberg

Friedenwald, J. S., Buschke, W., Scholz, R. O., Snell, A., and Moses, S. G. **Primary reaction of mustard with the corneal epithelium.** Bull. Johns Hopkins Hosp. **82**: 102-120, Feb., 1948.

The method used for exposing beef eyes to mustard vapor is described. There is an uptake of 3.3 micrograms of mustard per square cm. per minute and 8 percent of the mustard absorbed disappears per minute. The effective dose that produces minimal mitotic inhibition of rat cornea epithelium is 2.5×10^{-10} micrograms per cell. In the intact cell, glutathione is rel-

atively inaccessible to reaction with mustard.

A method is described to determine the electrical resistance of the cornea which is used as an index to the integrity of the cell membrane. Exposure to mustard does not result in any primary injury to the corneal epithelial cell membrane that is detectable by this method. Histologic studies show that tissue contaminated with mustard vapor has an increase in acidophilia, and that the region of the nuclear membrane and the basement membrane may be among the sites of primary mustard reaction. (8 figures, 5 tables.) H. C. Weinberg.

Friedenwald, J. S., Buschke, W., and Scholz, R. O. **Effects of mustard and nitrogen mustard on mitotic and wound healing activities of the corneal epithelium.** Bull. Johns Hopkins Hosp., **82**:148-160, Feb., 1948.

Slow inhibition of mitosis in the corneal epithelium of the rat exposed to mustard and nitrogen mustards is produced by doses too small to cause recognizable signs and symptoms of damage. Recovery from this inhibition occurs spontaneously but the inhibition can be made to last for several weeks by repeated instillations of the poison. During prolonged inhibition of mitosis the basal cells of the corneal epithelium increase in size.

Wounds of the corneal epithelium heal at a normal rate even in the absence of mitotic activity. Large doses of the toxic agent may slow down the whole mitotic cycle. Cells that are in mitosis at the time of exposure to the toxin in moderate doses will complete their division normally. Systemic administration of the poisons in MLD 50 dosage causes an inhibition of mitosis in corneal epithelium, bone marrow, and in intestinal mucosa. Inhibition of mitosis represents the lowest threshold effect so far recognized in the reaction of tissues to these poisons. (4 figures, 3 tables.) H. C. Weinberg.

Friedenwald, J. S., Buschke, W., and Moses, S. G. **Comparison of the effects of mustard, ultraviolet and X-radiation, and colchicine on the cornea.** Bull. Johns Hopkins Hosp. **82**:312-325, Feb., 1948.

A comparison of the effect on the cornea of mustard, ultraviolet and X-radiation and colchicine is given because of the similar effects of these substances in causing erythema, vesiculation, and ulceration of the skin. The administration of mustard causes the same effect on the viscera as that produced by exposure to any of the penetrating radiations. The inhibition of mitosis by ultraviolet and mustard show similar properties with those factors which cause nuclear fragmentation. In cells in which mitosis has been markedly inhibited by X-rays there was no nuclear fragmentation.

Loosening of corneal epithelium and the increased NPN content of the cornea after exposure to ultraviolet and mustard show no correlation with nuclear fragmentation or inhibition of a mitosis. The mechanism of loosening of the epithelium produced by colchicine may be different from that induced by mustard because of the production of increased NPN in the latter. (2 figures, 5 tables.) H. C. Weinberg.

Friedenwald, J. S., and Moses, S. G. **A mechanical device for the extraction of soluble compounds from the cornea and other tough tissues.** Bull. Johns Hopkins Hosp. **82**:350-352, Feb., 1948.

By simply squeezing a tissue that is placed in a vessel the extraction of soluble compounds is obtained as well as by the squeezing and shearing manipulation used in the grinding of the tissue in a mortar. A specially fitted plunger is at-

tached to a lever which reduces the amount of energy needed for this process. The device can be attached to a motor and six samples can be extracted simultaneously. (2 figures.) H. C. Weinberg.

Herrmann, Heinz. **The effect of histamine and related substances on the cohesion of the corneal epithelium.** Bull. Johns Hopkins Hosp. **82**:208-212, Feb., 1948.

Histamine in concentrations of 5 to 10×10^{-6} M. causes a loosening of the corneal epithelium. Chemically related natural bases such as histidine and tyramine have no such effect in concentrations up to 1×10^{-2} M. Adrenalin, ephedrine, and physostigmine cause a loosening of the corneal epithelium in concentrations of the order of 10^{-3} M.

This histamine effect develops slowly after some hours of incubation. The loosening produced by histamine occurs at the boundary between the basal epithelial cell layer and the overlying epithelium, not between the basal cells and the underlying stroma as is the case with other agents which were studied. (3 tables.) H. C. Weinberg.

Herrmann, H., and Hickman, F. H. **Loosening of the corneal epithelium after exposure to mustard.** Bull. Johns Hopkins Hosp. **82**:213-224, Feb., 1948.

In beef eyes a loss of cohesion between the corneal epithelium and the stroma is a prominent feature of mustard injury. This is not due to an inflammatory reaction nor is it a manifestation of death of the epithelial cells. This occurs before nuclear fragmentation. Anoxia and lowered temperatures aid tissues to recover to some degree from mustard injury. Exposure to adequate dosage of mustard causes corneal epithelium to slough with edema of the corneal stroma. The capillary endothelium of the conjunctiva likewise is affected. Petechial hemorrhages with packing of the red cells that cause obstruction to the circulation with a loss of plasma into the tissue are further manifestations of tissue damage.

Three phases of mustard injury are, the primary chemical reaction of mustard and tissue components which takes a few minutes, a second symptom-free period during which neither physiologic nor histopathologic changes are recognized and which may last up to twelve hours and the stage of recognizable symptoms of damage to the tissue. The pathologic mechanism of mustard injury suggests abnormal utilization of oxidative energy or the inhibition of some normally balancing reductive process. (8 tables.)

H. C. Weinberg.

Herrmann, H., and Hickman, F. H. **The adhesion of epithelium to stroma in the cornea.** Bull. Johns Hopkins Hosp. **82**:182-207, Feb., 1948.

A simple mechanical device is described for the quantitative estimation of the adhesion of the epithelium to the corneal stroma. Loosening of the corneal epithelium occurs after exposure to temperatures over 45 degrees C. or to freezing with subsequent incubation. Exposure to proteolytic enzymes also causes a loosening of the epithelium. The cohesion of the tissue is diminished on exposure to high concentrations of calcium chloride and sodium thiocyanate. No loosening of the epithelium was observed on altering the pH from 3 to 9.

No evidence was found to demonstrate the presence of an intercellular cement in the cornea. The adhesive forces may originate in the attraction of non-polar hydrocarbon residues in the boundary surface. The coherence surface may be a protein lipoid multilayer. The phenomenon of epithelial loosening in the cornea

is somewhat analogous to vesication in the skin. (10 tables, 6 figures.)

H. C. Weinberg.

Herrmann, H., and Hickman, F. H. **Exploratory studies on corneal metabolism.** Bull. Johns Hopkins Hosp. **82**:225-250, Feb., 1948.

The glycogen content of the epithelium of the beef cornea is like that of muscle, 1 percent. The phosphate fraction is the same as in the intestinal mucosa. The adenosine phosphate content is the same as in muscle also. The lactic acid content of epithelium is seven times as high as that of the blood. The glycogen content of the epithelium decreases 25 micrograms per hour by phosphorolysis and is not inhibited by iodoacetate. In the supravital cornea 1000 micrograms of glucose is metabolized in twelve hours but the process is completely inhibited by the addition of iodoacetate and is partially inhibited by fluoride. As long as glucose is supplied to the cornea the carbohydrate breakdown suffices for the maintenance of oxidations. In the excised cornea glycogen and lactate together do not provide sufficient substrate for the saturation of the oxidative mechanism. Lactate production from carbohydrate precursors takes place at a comparable rate in both the epithelium and in the stroma. Enzymatic oxidation is restricted to the epithelium, whereas the metabolism of the separated stroma is essentially anaerobic. (14 tables.)

H. C. Weinberg.

Herrmann, H., and Hickman, F. H. **The effect of mustard on some metabolic processes in the cornea.** Bull. Johns Hopkins Hosp. **82**:251-259, Feb., 1948.

Mustard injury to beef cornea inhibits utilization of lactate and causes a decrease in the oxygen uptake which is equivalent to that required for the complete combustion of the utilized lactate. The decline in oxygen uptake lags behind the decline in lactate consumption by one or more hours. The metabolic change preceeds the loosening of the corneal epithelium and is caused by the same dosage of mustard. Under anaerobiosis the loosening of corneal epithelium does not occur. After incubation of 15 hours the normal corneal lactate reservoir is depleted but the oxygen uptake rises instead of declining as it does after the utilization of lactate is inhibited by the mustard. Glucose and glycogen are utilized at a normal rate in mustard-treated corneas and their utilization is not associated with an extra accumulation of lactate except under anaerobiosis. In the cornea there is a special hydrogen transport system for a major part of the lactate oxidation which is damaged by mustard. A component of the system becomes oxidized and causes a loosening of the epithelium. (6 tables.)

H. C. Weinberg.

Herrmann, H., and Hickman, F. H. **Further experiments on corneal metabolism in respect to glucose and lactic acid.** Bull. Johns Hopkins Hosp. **82**:260-272, Feb., 1948.

Beef corneal epithelium consumes the lactate produced by the stroma and the rate of glucolysis in the stroma is influenced by the epithelium. The corneal epithelium exhibits a typical Pasteur effect in that glycogen is consumed more rapidly under anaerobic than under aerobic conditions. One quarter of the carbohydrate requirement of the epithelium is furnished in the form of lactate by the stroma.

Mustard injury causes a loss of cohesion between stroma and epithelium. The metabolic interaction between stroma and epithelium is connected with the mechanism for maintenance of tissue cohesion. Injury to this mechanism results in the loss of cohesion and in the loss of meta-

bolic interactions. The mechanism by which the epithelium consumes its endogenous stores of lactate is less susceptible to mustard injury than is the mechanism by which it acquires and consumes the lactate from the stroma. (10 tables.)

H. C. Weinberg.

Herrmann, H., and Hickman, F. H. **The consumption of pyruvate, acetoin, acetate, and butyrate by the cornea.** Bull. Johns Hopkins Hosp. **82**:273-286, Feb., 1948.

Pyruvate is consumed very rapidly by normal corneal tissue but very slowly by denuded corneal stroma. Anaerobiosis, or exposure to mustard, iodoacetate, fluoride or arsenite do not influence the consumption of pyruvate by the whole cornea. Half of the pyruvate can be recovered as lactate. One mole of carbon dioxide can be recovered for each four moles of pyruvate lost. Acetoin is consumed by the cornea during pyruvate consumption and is not inhibited by mustard. The very slow consumption of butyrate by the cornea is inhibited by exposure to mustard. (9 tables.)

H. C. Weinberg.

Herman, H., and Hickman, F. H. **The utilization of ribose and other pentoses by the cornea.** Bull. Johns Hopkins Hosp. **82**:287-294, Feb., 1948.

Beef cornea utilizes d-ribose and d-xylose one-tenth as fast as it utilizes glucose and one-half as fast as it utilizes lactate and glycogen. D-lyxose is used one-half times as fast as is d-ribose. There is no utilization of d-arabinose. An unknown intermediary compound is formed in the utilization of ribose. The breakdown of ribose in the cornea may involve phosphorylation.

Utilization of ribose is markedly inhibited by iodoacetate and fluoride, but is only slightly inhibited by mustard. In the presence of ribose the utilization of glycogen and of the lactate depot in the cornea is suppressed. (7 tables.)

H. C. Weinberg.

Herrmann, H., and Moses, S. G. **Studies on non-protein nitrogen in the cornea.** Bull. Johns Hopkins Hosp. **82**: 295-311, Feb., 1948.

The nonprotein nitrogen content of the beef cornea on prolonged incubation is quite constant. Exposure of a cornea to mustard causes a rise in extractable NPN from its protein. Six micrograms of mustard are taken up after ten minutes exposure by each square cm. of cornea. This is approximately 70 moles of nitrogen for each mole of mustard bound and approximately equal to the available SH groups to nitrogen in some proteins.

The oxidation of some proteins that react with mustard may increase their susceptibility to hydrolysis and may account for loosening of corneal epithelium after exposure to mustard. The NPN is increased before the loosening of epithelium appears. Anaerobic incubation of corneas exposed to mustard protects them against loss of cohesion.

Serine and lactate are normally consumed by the intact cornea but not by the denuded stroma. After exposure to mustard the concentration of serine rises in the stroma and falls in the epithelium. After injection of serine into the stroma and subsequent incubation the concentration in the epithelium was found to rise rapidly and to reach a level far higher than in the stroma. (11 tables.)

H. C. Weinberg.

Hughes, W. F., Jr. **The tolerance of rabbit cornea for various chemical substances.** Bull. Johns Hopkins Hosp. **82**: 338-349, Feb., 1948.

This paper consists of one large table in which data are compiled on the reac-

tions in 304 rabbit eyes when various chemicals are injected in varied concentrations. The chemicals include solvents, protein precipitating agents, mucoid dissolving agents, metals, arsenicals, oxidizing agents, quinones and hydroquinones, reducing agents, sulfhydril binding agents and dyes. (1 table.)

H. C. Weinberg.

Maumenee, A. E., and Scholz, R. O. **The histopathology of the ocular lesions produced by the sulfur and nitrogen mustards.** Bull. Johns Hopkins Hosp. **82**:121-147, Feb., 1948.

The conjunctiva of rabbits' eyes, when injured by mustard, shows edema of the stroma with a necrosis of the epithelium. In the capillaries there is a loss of endothelium, packing of the red cells and occlusion of the lumen. The corneal epithelium shows fragmentation of nuclei of the basal layer of cells which later become detached from the stroma, show pycnosis of the nuclei and slough.

Injury to the stroma is manifested early by edema of the posterior portion. The nuclei become pycnotic and may burst eight hours after injury. The polymorphonuclear leucocytes appear within twenty-four hours. The first evidence of damage to the corneal endothelium is a sloughing of the cells. Iris damage occurs especially after exposure to HN_2 and is first evidenced by a loss of the capillary endothelium with a packing of red cells and occlusion of the lumen. Repair of the conjunctival epithelium is accomplished by migration of cells from uninjured adjacent epithelium. Macrophages remove the debris. Occasionally "pearly white" avascular areas are observed as well as areas with new capillaries. The corneal epithelium is repaired by a sliding of adjacent uninjured corneal epithelial cells. In the corneal stroma polymorphonuclear leucocytes remove the debris and large macrophages are transformed into keratoblasts. Scarring with blood vessels appears if the injury is severe. The repair of the endothelium begins ten days after injury by a migration of uninjured cells. Repair of the iris is essentially the same as that of conjunctiva. (26 figures, 1 graph.) H. C. Weinberg.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Angius, Tullio. **Research concerning accommodation.** Rassegna ital. d'ottal. **16**:460-466, Nov.-Dec., 1947.

Angius shows that the accommodative power varies with somatic conditions and with age. As much as three diopters difference may be found in normal individuals. His studies of the 19 to 26-year age group show the same curve as that of Duane. A plea is made for more exact study of the accommodation and for more precise instruments of investigation.

Eugene M. Blake.

Belmonte Gonzales, N. **Observations on corneal astigmatism.** Arch. Soc. oftal. hispano-am. **7**:1169-1172, Dec., 1947.

In fitting contact lenses Belmonte was impressed with the fact that they usually correct the total astigmatism of the eye. This raises a question as to the existence of a lenticular astigmatism, to which the discrepancy between ophthalmometric data and the total astigmatism of the eye is usually attributed. The sources of error in ophthalmometric measurements are discussed, and it is suggested that an ophthalmometer without these defects could be constructed. In the meantime the author finds the bicylindric method of Marquez the most accurate means for determining the ocular astigmatism.

Ray K. Daily.

Blanchi, G. **A new explanation for the variation in refraction in diabetics.**

Rassegna ital. d'ottal. **16**:433-440, Nov.-Dec., 1947.

The various theories to explain the sudden changes in refraction in patients with diabetes are reviewed. The author suggests a new explanation, namely an axial displacement of the lens, and shows by some rather complex formulas how it is possible for this to occur. Two cases of rapidly developing hypermetropia are reported. Eugene M. Blake.

Dascalopoulos, N. **Acquired inversed astigmatism.** Ann. d'ocul., **180**:688-695, Nov., 1947.

Operations which alter the corneal curvature all produce an inverse corneal deformity. This occurs at the point of least thickness and in the direction of least resistance. Inverse astigmatism also occurs in myopia, glaucoma and hyperthyroidism and with chalazion and scleral keratitis. The corneal curvature basically depends upon the ocular tension, the pull of the extraocular muscles and the supporting structures, namely the orbit behind, and the lids anteriorly. Any condition which disturbs the equilibrium of these forces vertically or horizontally may produce inverse astigmatism. Chas. A. Bahn.

Hardy, L. H., Rand, G., and Rittler, M. C. **A screening test for defective red-green vision.** Arch. Ophth. **38**:442-449, Oct., 1947.

The authors have selected 18 plates from the Pseudo-Isochromatic Plates for Testing Color Perception, compiled in 1940 by the American Optical Company. When these 18 plates are used under standard conditions of illumination, they serve as a satisfactory screening test for red-green defects. It is neither qualitative nor quantitative; that is, it does not classify the type of deficiency in red-green vision, or the degree of deficiency. No provision is made for detection of the rare forms of defective blue-yellow vision. Using these plates, an incorrect response to five or more plates indicates defective red-green vision.

John C. Long.

Latorre, S. **High transitory corneal astigmatism in a case of sphenoidal ethmoiditis.** Arch. Soc. oftal. hispano-am. **7**:1009-1012, Oct., 1947.

In the course of an orbital cellulitis, an extension of an ethmoidal sinusitis, a nine-year-old child developed an astigmatism of 3.50 diopters. The astigmatism disappeared after recovery, indicating that it was caused by compression of the eyeball. Another patient could temporarily abolish a corneal astigmatism of four diopters by pressure of the upper lid against the cornea. Ray K. Daily.

Littwin, Charles. **Use of one contact lens in correction of high unilateral myopia.** Arch. Ophth. **38**:259-260, Aug., 1947.

In a patient with 13 diopters of myopia in one eye and only one quarter diopter of myoptic astigmatism in the other fusion and comfort could be obtained by the use of iseikonic lenses only with the eyes in the primary position. When a contact lens was prescribed for the myopic eye, comfortable binocular vision could be obtained in all directions of gaze.

John C. Long.

Mathewson, W. R. **Rapid retinoscopy.** Med. Press **219**:101-102, Feb. 4, 1948.

The author describes a rapid method of performing retinoscopy. It is a modification of a procedure described by Harrison Butler and the principal instruments are a Purvis Streak retinoscope and a Hamblin retinoscopy lens rack. The

work is done at a distance that is at slightly greater than one-half meter.

Francis M. Crage.

Matteucci, P., and Toselli, C. **The accommodation time in paralysis of the cervical sympathetic and in accommodative spasm.** Rassegna ital. d'ottal. **16**:407-417, Nov.-Dec., 1947.

In the patients affected by Horner's syndrome the paralysis of the sympathetic did not modify the speed of accommodation nor the amplitude. Spasm of accommodation provoked a prolongation of this act, for distance or for near, or for both. Astigmatic and hyperopic eyes always showed an increased power of accommodation. Low grade myopes and emmetropes showed little variation in the accommodative time for near.

Eugene M. Blake.

Pacalin, G. **The diploscope in the prescription of glasses.** Ann. d'ocul. **180**:696-701, Nov., 1947.

The diploscope of Remy has been much more widely adopted in France than elsewhere. It is based on the principle that a person looking through two apertures which correspond with the binocular line of fixation will see three horizontal letters if properly distanced and sized. They will be in their regular order and correctly placed if binocular vision is normal; but, if abnormal, the letters will either be doubled, omitted or improperly arranged. The diploscope is therefore used as a simulation test to educate binocular vision, verify the respective sizes of the retinal images in each eye, verify the accuracy of refractive corrections especially in anisometropia, to detect ocular motor and convergence disequilibrium and abnormal levelling of the two eyes. If convergence is insufficient or relative exophoria exists, crossed diplopia will result. With convergence excess or relative esophoria there is homonymous diplopia.

Chas. A. Bahn.

Radnót, Magda. **Data on the occurrence of calcification in the eye tissues.** Brit. J. Ophth. **32**:47-54, Jan., 1948.

The zonular opacity seen in the region of the palpebral aperture as an opaque band is a calcified degeneration of the layers under the epithelium. It occurs as an occupational disease in hat makers and is caused by the deposition of rabbit hairs on the cornea. A second type is primary in otherwise healthy eyes and a third is secondary to ocular damage; the latter usually follows severe uveitis. In this report, 12 such cases of corneal calcification are presented in each of which there was also lime in other tissues of the eye. These deposits occurred in the lens, choroid, detached retina, optic nerve and in old chorioretinal scars. In some of them actual formation of bone was found. (14 figures.)

Morris Kaplan.

Ronchi, V. **Recent developments in the theory of form and color vision.** Arch. Soc. oftal. hispano-am. **7**:959-1000, Oct., 1947.

This is a general review of physiologic optics, from the time of Rayleigh's investigations. The author's work on the resolution of visual images is based on the data obtained from the study of the resolution of photographic emulsions. He applies Hecht's photochemical theory to color vision as well as to form vision, by assuming that the impulses of form and color vision differ in frequency. He demonstrates the similarity between retinal physiology and the phenomena of photographic emulsions, points out the possibility of transformation of visual radiations into acoustic stimuli, and advances a unitarian theory of the mechanism of sensory functions.

Ray K. Daily.

Vila-Coro, A. **Fukala's method of treatment of high myopia.** Arch. Soc. oftal. hispano-am. **7**:1013-1026, Oct., 1947.

Vila-Coro is an enthusiastic advocate of lens extraction for the arrest of progressive myopia. Discission in patients under 15 years of age, discission followed by a linear extraction in adults with transparent lenses, and intracapsular suction extraction in adults with opacities in the lens are the procedures advocated. The indications for the operation are progressive myopia of not less than ten diopters, and myopia in patients over 40 years of age who have incipient lenticular opacities. Vitreous, choroidal, or retinal hemorrhages are regarded as contraindications to surgery. Degenerative lesions which do not involve the macula do not contraindicate the operation. To arrest the progress of myopia, and to improve ocular nutrition, both eyes should be operated, with an interval of six to eight months between the operations.

Ray K. Daily.

5

DIAGNOSIS AND THERAPY

Azzolini, U., and Pascucci, F. **Streptomycin therapy in Reiter's infections.** Giorn. ital. di oftal. **1**:14-22, Jan.-Feb., 1948.

Streptomycin was used in the treatment of three men afflicted with Reiter's disease of long duration. There was marked improvement in the urethral and conjunctival condition, but none in that of the joints. Francis P. Guida.

Dollfus, M. A., Barrois, P., and de Grénédan du Plessis. **Tissue therapy of Filatov.** Ann. d'ocul. **180**:681-687, Nov., 1947.

The details of the Filatov treatment have been so repeatedly recently abstracted or described in this journal that the preparations of the placenta and its extracts, dead skin, cod liver oil, and details are here omitted. The author restates the conclusions of the article by Filatov and Verbitskaia in which the progress is measured in such small units as to be understood only by God, and from which one draws the conclusions that figures don't lie, but—. The authors report on two cases of retinal pigmentary degeneration treated by the Filatov method, one of which was possibly functionally improved. Six cases of high myopia were also treated without any appreciable improvement functionally or structurally. Chas. A. Bahn.

Dubois-Poulsen, A. **A new instrument in surgery of the face: the dermatome.** Ann. d'ocul. **180**:664-671, Nov., 1947.

The dermatome of Padgett which was observed in action by the author during the recent war has been modified by him, making it smaller. This instrument consists essentially of a rectangular, metal frame with a hand grip on one end. Attached to the base is a razor-like blade with a micrometer adjustment. This adjustment is regulated by a swinging arm. Thus the section of the skin is accurately regulated, and the swinging arm propels the excised skin forward to facilitate its preservation. This instrument is considered of signal service especially in entropion, and also in orbitoplasties. In the latter the skin is accurately fixed to the free lid border and the graft is held in place by an acryllic mould.

Chas. A. Bahn.

Greeves, R. A. **Technical ophthalmological details.** Tr. Ophth. Soc. U. Kingdom **65**:341-346, 1945.

The paralysis of the orbicularis muscle is obtained by blocking the facial nerve in the cheek. Peripheral iridectomy has been discarded for the complete one. The

trephine should be applied slightly obliquely and the lateral hinge finally separated with scissors to prevent its loss into the anterior chamber. Greeves advises against the use of a miotic immediately following a trephine operation as a quiet chronic iritis may follow and the pupil become firmly adherent posteriorly. Beulah Cushman.

Grignolo, Antonio. **Clinical and experimental use of streptomycin in ocular tuberculosis.** Minerva med. **39**:16-20, Jan., 1948.

Grignolo inoculated the anterior chamber of the eyes of 33 guinea pigs with a virulent strain of human tubercle bacilli. One group was used as a control and one received full doses of streptomycin six days after the inoculation. Another group was treated with streptomycin 15 days after inoculation, and the fourth with streptomycin and sulfone. The group treated early showed marked inhibition of the tuberculous process. Those whose treatment was commenced on the fifteenth day were no better than the control group, thus demonstrating the advantage of early treatment, at least in the experimental animal.

Seven patients with ocular tuberculosis were treated with streptomycin. In two with choroiditis there was a favorable response and in two with anterior uveitis no improvement. One patient with miliary pulmonary disease and choroidal foci was greatly benefited, whereas two with phlyctenular keratitis were not improved.

Eugene M. Blake.

Grignolo, Antonio. **Clinical research on the employment of streptomycin in non-tuberculous ocular lesions.** Minerva med. **1**:271-275, March 17, 1948.

In 72 patients streptomycin was administered by instillation, unguent, ocular baths, subconjunctival injection and injection into the anterior chamber. The dosage was that of tolerance, avoiding the state of streptomycin resistance. Good results were obtained in blepharitis ulcerosa, acute staphlyococcus and Koch-Weeks conjunctivitis, but no better than with penicillin. Gonococcic infections improved under treatment with streptomycin but were not cured. In one case of inclusion conjunctivitis, the inclusion bodies disappeared rapidly. In 12 cases of florid trachoma the conjunctival sac was rapidly sterilized, but the nodules were unaffected. No improvement was noted in the treatment of corneal ulcerations due to pneumococcus, staphylococcus or streptococcus, nor in herpes, epidemic keratoconjunctivitis or dacryocystitis. Streptomycin appeared to be effective against b. coli, b. Friedländer, b. proteus and a few other organisms.

Eugene M. Blake.

Iliff, C. E. **Beta irradiation in ophthalmology.** Arch. Ophth. **38**:415-441, Oct., 1947.

The majority of reports on radiation therapy of the eyes are concerned with gamma rays. Beta rays possess the same qualities as other rays in the power of selective tissue destruction, but in contrast to gamma rays, they penetrate about 3 mm. of tissue only. In no case in which beta ray therapy has been used has damage to the lens been observed. Treatment is carried out with a special radon applicator. Excellent results are reported from the use of beta irradiations in vernal conjunctivitis, tuberculosis of the anterior ocular segment and in small benign tumors of the anterior segment. The agent is ideal for preventing superficial vascularization after keratectomy and keratoplasty. Granulation tissue, conjunctival flaps and pterygiums can be made avascular with contact therapy. Encouraging results are obtained in acne rosacea

keratitis. Corneal dystrophies, sarcoid, lupus erythematosus, pemphigus, punctate keratitis and corneal ulcers due to pyogenic infections are not benefited by beta rays, and may be made worse by their use. John C. Long.

López Enríquez, M. **Retinography with a short focus camera.** Arch. Soc. oftal. hispano-am. **7**:1038-1041, Oct., 1947.

To convert the large Gullstrand ophthalmoscope into a fundus camera, Enríquez increased the illumination, by using a stronger nitra lamp, and attached a Leica camera to the ocular by means of a collar. (2 illustrations, 4 fundus photographs.) Ray K. Daily.

Morón-Salas, José. **For corneal examination.** Arch. Soc. oftal. hispano-am. **8**:90-92, Jan., 1948.

Morón-Salas devised a mounting to hold a twenty-diopter lens and a piece of blue glass. This device serves as a substitute for an ultraviolet light, to intensify fluorescin stained corneal defects, to demonstrate corneal lesions such as keratitis filamentosa, and to check on the fit of contact lenses. It does not bring out the fluorescence of the lenticular cortex. (3 illustrations.) Ray K. Daily.

Traquair, H. M. **Pre-operative and post-operative treatment in eye operations.** Tr. Ophth. Soc. U. Kingdom **65**: 356-365, 1945.

This paper deals with the various circumstances and factors with which the surgeon is concerned other than the operation itself. Psychological disturbance is much more common than physical disease, therefore, pre-operative treatment is largely psychological. The patient should be encouraged to take the operation in his stride. Local preparatory measures should be reduced to a minimum. After operation the patient may walk back to bed or be taken on a cart if the operating theatre is some distance away. The patient should be comfortable in bed after operation especially after cataract operations. Hemorrhage into the anterior chamber is frequently due to disturbed sleep. The patient's personal habit of taking cathartics should not be changed because of the operation. Postoperative embolism may be reduced by having the patients move the legs freely several times a day for about five minutes. Dark glasses are recommended and the patients are warned not to rub the face with a towel for six weeks, until the wound is well healed.

Beulah Cushman.

6

OCULAR MOTILITY

Adler, F. H. **Some confusing factors in the diagnosis of paralysis of the vertically acting muscles.** Am. J. Ophth. **31**: 387-397, April, 1948. (17 figures.)

Aguilar y Menoz, José. **Diagnosis and treatment of heterophoria with the heteroscope.** Arch. Soc. oftal. hispano-am. **7**: 1027-1037, Oct., 1947.

Aguilar's heteroscope is devised on the principle of Remy's diploscope and is used in a similar manner. The instructions for its use are given in detail. It is urged that orthoptic training should be continued to the point of simultaneous perception before surgery is done for strabismus, and to the point of binocular vision after it, in order to avoid a recurrence. (16 illustrations.)

Ray K. Daily.

Arriago Cantullera, José. **Postanesthetic ocular paralysis.** Arch. Soc. oftal. hispano-am. **7**:1199-1208, Dec., 1947.

The literature is reviewed, and three cases of ocular paralysis are reported,

which followed the injection of 6 cgr. of estovain between the first and second lumbar vertebrae. In the first patient paralysis came on in both eyes one week after the operation. The patient made a complete recovery under intensive therapy with vitamin B. Two others are still under treatment. The various theories on the pathogenesis of this accident are discussed and no new viewpoints are presented. Ray K. Daily.

Hudelo, A. **Syndrome of Hertwig Magendie.** Ann. d'ocul. **180**:702-705, Nov., 1947.

In a nine-year-old boy, disassociation of gaze and voluntary ocular movements was observed. He had defective dental articulation. Vision in the right eye was 0.9 and in the left, 0.6, and there was slight strabismus. In this syndrome vertical disassociation of the visual axes is present with proprioceptive inadequacy. Chas. A. Bahn.

Latorre, S. **Sensomotor orbital postoperative ophthalmoplegia.** Arch. Soc. oftal. hispano-am. **7**:1173-1175, Dec., 1947.

Latorre reports a very unusual occurrence after a retrobular injection of 4-percent novocain solution used in the course of removal of a traumatic cataract. At the end of the operation the levator of the upper lid was paralyzed. At the end of 48 hours when the bandage was removed, the patient presented a picture of complete ptosis, paresis of the inferior and internal recti, and anesthesia of the skin innervated by the frontal and external nasal nerves. There was slight anesthesia of the upper half of the cornea. There was no hematoma in the orbit. Intensive vitamin B therapy brought about an improvement in two weeks, and a complete cure in two months. Ray K. Daily.

Lisman, J. V. **A triple-armed suture for resections.** Am. J. Ophth. **31**:466, April, 1948. (2 figures.)

Orzalesi, F. **Heredofamilial degeneration of the cornea: histological report.** Rassegna ital. d'ottal. **16**:393-406, Nov.-Dec., 1947.

An instance of corneal degeneration in an 8-year-old girl is carefully described by the author. Although the lesion probably belongs in the heredofamilial group, there were atypical features. The principal alterations were situated exclusively in the anterior layers of the parenchyma and consisted of small foci of destruction of the corneal lamellae and spaces, either empty or filled with corneal detritus. The basal layer of epithelium showed vascularization, especially over the stromal foci. Bowman's membrane presented defects in some places.

The author suggests that to the three types of corneal degeneration described (Groenow's nodular, Dimmer's reticular and Feher's granular) there be added a fourth type. More intensive study of the fatty elements could perhaps clarify the picture. (5 figures.) Eugene M. Blake.

Scobee, R. G., and Green, E. L. **Relationships between lateral heterophoria, prism vergence, and the near point of convergence.** Am. J. Ophth. **31**:427-441, April, 1948. (8 figures, 7 references.)

7

CONJUNCTIVA, CORNEA, SCLERA

Allenic. **An interesting case of conjunctivitis sicca.** Ann. d'ocul. **180**:706-707, Nov., 1947.

A woman, aged 62 years, after a short attack of unexplained fever developed a diminution of tears and salivary secretion with parotid inflammation. After approximately a week, all the symptoms subsided. The author associates these symp-

toms with the syndrome of Sjögren which may be due to parasympathetic hypofunction involving the vagus nerve.

Chas. A. Bahn.

Azzolini, U. **A report of mycosis of the conjunctivia in self-pitying individual.** Riv. di oftal. **2**:112-118, March-April, 1947.

In a young self-pitying woman afflicted for eight years with a unilateral chronic conjunctivitis of the lower fornix, myces of the Macrosporium were found in the secretions. The canaliculus was not involved. It was decided that the lesion was due to a self-induced injury secondarily infected by the fungus.

Francis P. Guida.

Bodian, Martin. **Trachoma.** Arch. Ophth. **38**:450-460, Oct., 1947.

Of 100 natives working at an American Army base in Fiji 22 percent had clinical evidence of active trachoma and 42 percent harbored Prowazek-Halberstädter inclusions in the conjunctival epithelium. Of the natives with clinical trachoma, 68 percent showed typical conjunctival inclusions. Inclusion bodies morphologically indistinguishable from those observed in frankly trachomatous patients were found in 34.6 percent of the natives with no clinical evidence of the disease. This observation suggests the existence of a carrier state in trachoma. Transmissibility and pathogenicity of the virus of trachoma from these patients still await demonstration. Fifty American soldiers based in Fiji for from one to three years failed to show clinical trachoma or inclusion bodies.

John C. Long.

Casari, G. F. **Histological study of the conjunctival nodules in erythema nodosa.** Rassegna ital. d'ottal. **16**:467-472, Nov.-Dec., 1947.

Erythema nodosa developed in a 14-year-old girl whose general health was excellent, but who had previously had a dry pleurisy. An eruption appeared at the inner and outer angles of the bulbar conjunctiva of each eye. Microscopic examination showed no changes in the pavement epithelium, below which was fibrous and hyaline tissue, with edema and necrosis. There were scattered areas of perivascular lymphocytic infiltration. There were some epithelioid elements with fragmentation of the nuclei. The ocular condition was believed to be a para-allergic manifestation of tuberculosis.

Eugene M. Blake.

Castroviejo, R. **Keratoplasty and keratectomy—indications and contraindications.** Ann. d'ocul. **180**:577-586, Oct., 1947.

Generally speaking, keratoplasty is preferable to keratectomy because of better visual results. Active inflammation and glaucoma are contraindications to either operation. Eyes should be free from inflammation for at least six months, and hypertension should be adequately controlled before any surgical treatment can be considered.

Three groups of patients are analyzed. 1. In the most favorable group are those with slight corneal opacities, especially those due to keratoconus and interstitial keratitis. 2. In the less favorable group are most cases of corneal dystrophy without vascularization but with normal stroma and endothelium. Included are the types of Fleisher, Haab-Dimmer, Groenow, and Salzman. 3. In the least favorable group the patients have more extensive cicatrization especially with vascularization, extensive pupillary zone involvements, adherent leucomas, less favorable dystrophies, severe thermal and chemical burns, corneal opacities in aphakic and amblyopic eyes, calcareous deposits and other degenerative diseases such as pemphigus. Preliminary radium

therapy to reduce vascularization, partial or complete keratectomy, iridectomy and other measures may be advisable adjuncts. The surgical risks involved must be justified. Chas. A. Bahn.

De Laet, H. **The profile of circular corneal grafts.** Ann. d'ocul. **180**:629-638, Oct., 1947.

Is especially the marginal profile of corneal trephined grafts modified by differences of curvature, pressure and tension? Experiments on pigs' eyes, human eyes, and on rubber tubes made to simulate the human cornea, were carried on to elucidate the physical laws involved as well as their practical applications in both the donor and enucleated corneas. Theoretically some deformity does exist, but practically the construction of the human cornea overcomes this tendency. The lamellar structure of the stroma with its elastic fibers sandwiched between two non-elastic membranes, those of Bowman and Descemet, prevents distortion of the disc. The author, however, favors the removal of the disc from the donor cornea by a stamping rather than a sawing motion of the trephine. For this purpose the donor cornea is placed on a corresponding plastic disc. Chas. A. Bahn.

Dor, Louis. **Chronic conjunctivitis in cholesterolemia.** Ann. d'ocul. **180**:708-709, Nov., 1947.

The association of some chronic conjunctivitides and palpebral eczemas with quantitative and qualitative lipid abnormalities is briefly discussed. The administration of a drug, chophytol, which apparently is a choline preparation, was in some patients followed by more rapid improvement than otherwise might have been expected. Chas. A. Bahn.

Heyrowsky, K. **A case of vicarious bleeding into the conjunctiva and bloody tears.** Wien. klin. Wchnschr. **59**:702-704, Oct. 24, 1947.

A 26 year old woman, the fourth one presenting the same symptoms, had a polyglandular disturbance, genital hypoplasia and recurrent vicarious bleeding into the eye. Her female ancestral tree is presented and other members of her family were found to have the same vicarious bleeding. The patient was given various hormonal extracts, her medical condition was improved by general regime and after 1½ years of treatment the patient had no recurrences.

Theodore M. Shapira.

Hughes, E. L. **Local sulphonamide therapy of dendritic ulcer.** Brit. J. Ophth. **32**:43-46, Jan., 1948.

Local sulphonamide therapy in the form of albucid ointment was used in 16 consecutive cases of dendritic ulcer. Thrice daily 6-percent ointment was instilled and the entire corneal surface was painted daily with 30-percent solution. Of the 16 eyes, 12 responded well. Improvement seemed to be directly proportional to corneal sensitivity; the greater the loss of sensitivity, the less favorable was the result. Those that did respond well were clinically cured within eight days.

Morris Kaplan.

Kettesy, A. **On results obtained by total conjunctival hooding of the cornea for serpiginous ulcer.** Brit. J. Ophth. **32**: 36-43, Jan., 1948.

Total hooding of the cornea by a conjunctival flap was first introduced for the treatment of Mooren's ulcer and in the author's hands was so very satisfactory that in his clinic it has become routine treatment for serpiginous ulcer as well. In this report 56 consecutive cases of serpiginous ulcer so treated are described and are compared to twice as many treated in the ordinary, routine

way which did not include the use of penicillin. The results were uniformly satisfactory. The conjunctiva is undermined above, is brought down and is sutured to the normally attached conjunctiva below the cornea. The procedure failed only when the sutures were placed across the cornea. The hood was left in place for six months and then removed with scissors. In all cases pain ceased immediately. Visual improvement was well over twice that obtained by other measures and the hospital stay was reduced from 14 days to six.

Morris Kaplan.

Larsson, S. **Treatment of perforated corneal ulcer by autoplastic scleral transplantation.** Brit. J. Ophth. **32**:54-57, Jan., 1948.

Soon after successful removal of a retrobular hemangioma in a seven-year-old child a severe corneal ulcer developed because of lagophthalmos. Despite all routine treatment, and four attempts to cover it with a conjunctival flap, the ulcer persisted, became larger and developed a large descemetocele. A scleral button was removed by means of an ordinary punch, the ulcer edges were excised, the button placed in the opening and a conjunctival flap covered over it. The result was very satisfactory; the button remained in place, the ulcer remained closed, the flap persisted and the vision improved from 0.2 to 0.5.

Morris Kaplan.

Magitot, A. **The transparency of corneal grafts.** Ann. d'ocul. **180**:619-628, Oct., 1947.

Corneal transplantation is not new; in animals, 125 years old; in humans, only 65 years. The results of 100 transplants were reported by Elschnig in 1925, and but few basic improvements have been made since. American exuberance and over-dramatization of keratoplasty, especially in the popular press, was editorially commented upon recently in this journal. Corneal healing and transparency is largely dependent upon the normalcy of the epithelium and the endothelium. Rapid epithelial reproduction is apparently due to an extension of conjunctival proliferation. But little is known concerning the nutrition and replacement of the stromal cells. Although the cornea and sclera are similar anatomically, one is transparent but the other is opaque. This transparency is largely due to a hydrophile gel which exists both in colloid and crystalline form. The refractive index is of less importance in corneal transparency than the hydrophile gel. Under normal conditions oxygen passes through the cornea into the anterior chamber and carbon dioxide passes in the reverse direction. In the presence of toxic agents, cations are retained in the cornea by both systems, affecting both corneal permeability and transparency. Anaesthetic corneas are predisposed to opaque grafts, which means that nervous regulation is involved. Each epithelial cell is attached to a nerve filament. It has long been known that partial keratectomies with a healthy bed frequently retain their transparency. In pemphigus, however, the stroma remains clear but the epithelium soon becomes opaque. The corneal metabolic rate is high; many wearers of contact glasses observe Fick's phenomenon. Autoplastic grafts are more frequently successful than homoplastic. Of the latter, approximately 20 percent are incompatible with the cornea of the host and therefore doomed. Heteroplastic grafts are almost uniformly unsuccessful in man. Corneas for grafts are best preserved at 4 to 7 degrees C. They may remain vital for fifteen years at this temperature. The best preservative media are hemolized serum and cerebrospinal

fluid. The performance of keratoplasty is not more difficult than cataract extraction, and it must be undertaken with the same sense of its moral and medical responsibility. Chas. A. Bahn.

Michtner, H. **Lesions in corneal epithelium caused by spraying of lacquer.** Proc. Ophth. Soc. Vienna p. 36, Jan. 18, 1943.

Some months after having started working as a lacquer sprayer a man developed pain in both eyes and headache. Slitlamp examination revealed minute whitish opacities in the corneal epithelium which did not impair the vision. After a change of occupation the corneal lesions disappeared completely.

F. Nelson.

Neame, Humphrey. **A rare case of bilateral macular keratitis with cyclitis, affecting all layers of the cornea. Comparisons with corneal lesions present in some virus infections.** Tr. Ophth. Soc. U. Kingdom **65**:328-340, 1945.

The eyes of a patient with bilateral macular keratitis, two with nummular keratitis and two with disciform keratitis are described. The opacities were numerous but countable and the depth variable. The size of the spots varied from 0.5 to 1 mm. The disappearance of the spots was complete except in the severe cases of disciform and zoster keratitis.

The author concludes that nummular keratitis lies morphologically between superficial punctate keratitis and disciform keratitis and that the one case he presented forms a link between superficial punctate keratitis and nummular keratitis. (5 figures.)

Beulah Cushman.

Nizetic, Z. **Visual results after corneal grafts.** Ann. d'ocul. **180**:596-603, Oct., 1947.

After a brief review of the subject one sees this statement: good vision in one eye usually means that keratoplasty is superfluous on the other. Twelve personal cases of operation performed during a period of fifteen years are reported with observation from six months to eleven years afterward. The keratoplasties were necessitated by trachoma, serpentic ulcer and parenchymatous keratitis. Vision ranged from 0.1 to 1. Chas. A. Bahn.

Offret, G. **The biologic problems of keratoplasty.** Ann. d'ocul. **180**:613-618, Oct., 1947.

The successes reported in homografts range from 40 to 85 percent. Preservation of the cornea at 4°C. preserves sufficient vitality for replacement surgery. Corneal transplantation somewhat resembles tendon transplantation. Both are essentially mesodermal and their basic structures are not replaced after transplantation. The epithelium and endothelium are of vital importance in maintaining transparency in keratoplastic grafts. Allergy and immunity play a more important part in success than has been realized. Allergic toxic reactions of the donor implant to bacterial and other toxins of the host are relatively frequent. Keratoblasts in the donor cornea succumb within three days and are replaced by those of the recipient cornea, which possibly arise from epithelial cells. Cicatricial tissue and new blood vessels interfere most with the vitality of the graft. In some types of hereditary dystrophies the donor graft is not poisoned by the cornea of the host. The early life of the graft depends largely upon the diastatic activity of the stromal cells. Keratoplasty has been used therapeutically in some acute corneal diseases.

Chas. A. Bahn.

Paufique, L. **Defense of non-perforating corneal grafts.** Ann. d'ocul. **180**:587-595, Oct., 1947.

This type of operation is advisable largely in corneal opacities, especially bilateral, which cause practical blindness. These include: 1. inflammatory diseases such as tuberculosis, syphilis, rosacea, trachoma, severe thermal and chemical burns; 2. degenerative diseases, especially severe keratoconus, recurrent progressive pterygium, and severe dystrophies. Optical iridectomy may be a preliminary or subsequent adjunct. The underlying opaque corneal tissue forms a protective wall for the intraocular contents and does not reduce vision as much as is generally supposed. Obviously the results are not brilliant. The 60 cases reported were unfavorable surgical risks. Patients must be carefully selected for this type of surgery which must be expertly performed. Chas. A. Bahn.

Somerset, E. J. and Sen, K. **Intra-ocular foreign bodies. An account of military cases from the Burma-Assam front.** Brit. J. Ophth. **32**:13-23, Jan., 1948.

Techniques and results of care in 29 selected cases of intraocular foreign body are presented. (These cases were evacuated by air personally by the abstractor from the Burma front into India). Each patient selected had a retained foreign body within the eyeball and each retained fair light perception. Most patients were seen several weks after the injury and most had received routine administration of sulpha drugs by mouth when first injured. Fifteen were British and 14 were Indian and the latter tended to react much less to surgery or injury than the former. There were no cases of serious infection and no cases of sympathetic ophthalmia. Morris Kaplan.

Sourdille, G. P. **Perforating partial grafts of the cornea: their technique and results.** Ann. d'ocul. **180**:604-612, Oct., 1947.

The donor eye after enucleation is placed in paraffin oil, kept at a temperature of 4°C. and finally washed with serum. Two sized trephines are used: in the donor eye, 5.1 mm.; in the recipient eye, 5 mm. Descemet's membrane should not be traumatized. A lid suture is used, the eye is bandaged 17 days, and examined after five days.

The success of the transplant depends partly on the normality of the donor cornea and especially the uveal tract. Centrally located opacities with sharply limited margins in the recipient eye are a good omen. Increased intraocular tension following the operation as well as vascularization are bad omens, as are also slight vitreous loss, mobility of the graft adhesions between cornea and iris, and lowered vitality of the surrounding cornea, as in severe alkaline burns. In one case sympathetic ophthalmia was observed. Chas. A. Bahn.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Barany, E. H. **The mode of entrance of sodium into the aqueous humour.** Acta Physiol. Scandinav. **13**:55-61, 1947.

Radio-active sodium chloride in solution was injected intraperitoneally into anesthetized rabbits. Studies of aspirated aqueous were made on animals whose bloodpressure in the ophthalmic and retinal artery was lowered by carotid clamping and on control animals whose arteries were not clamped.,

Those with obstructed carotids should have shown a lowering of the accumulation of sodium in the chamber if ultrafiltration affected the dynamics of passage of the sodium. Since the rate accumulation of sodium was not greatly affected by the clamping and the subsequent lowering of arterial pressure, the author believes that the process involved

in the passage of the sodium was not one of filtration but of secretion.

Francis M. Crage.

Barany, E. H. **The relative importance of ultrafiltration and secretion in the formation of aqueous humour as revealed by the influence of arterial blood pressure on the osmotic pressure of the aqueous.** Acta Physiol. Scandinav. **13**:81-86, 1947.

It has been shown that clamping the carotid artery reduces the rate of ultrafiltration by reducing the filtering pressure in an eye. If untrafiltration played an important role in the formation of the aqueous humour then one should expect that clamping a carotid would influence the osmotic pressure of the aqueous.

Osmotic pressure studies were made on aqueous aspirated from each of the two eyes of rabbits in which one carotid was clamped off. After the blood pressure of the eye on the clamped side had been kept at 30 mm. Hg below normal for one and one-half to two hours no significant difference in osmotic pressure of the aqueous was detected between the two eyes. The amount of filtered aqueous therefore could not be considerable. This favors the theory of secretory production of the aqueous humor.

Francis M. Crage.

Laval, Joseph. **Hyaline membrane of the iris. Report of two cases.** Am. J. Ophth. **31**:461-463, April, 1948. (5 figures.)

Lowenstein, Arnold. **The sympathizing eye.** Tr. Ophth. Soc. U. Kingdom **65**:219-231, 1945.

The author presents the findings in the exciting and sympathizing eyes in two cases. He stressed the findings of corneal epithelial edema, the digestive power of granulomatous tissue towards the glass membranes, the existence of huge macrophages filled with lens matter in the anterior chamber and the great mass of eosinophiles, the granulomatous tissue in the vitreous with epithelioid and giant cells especially in the pre-retinal region. The huge perivascular retinal infiltrates contained many plasma cells and plasma cell rows lined the internal limiting membrane. The histologic findings refute the allergic theory of sympathetic ophthalmia. The provoked allergic uveitis resembled endopthalmitis and not sympathetic ophthalmia.

The saprophytic virus of the conjunctiva may be transferred by a perforating wound into the contents of the globe. If the strain is neurotropic the alleged virus creeps along the ciliary or optic nerve to the brain. In cases of sympathetic ophthalmia it migrates along the nerves to the other eye. The infection of the other eye is patch-like. The repeated reabsorption of the virus creates allergy with release of an H-substance where the nerve endings enter the tissue of the second eye.

This assumption would explain: 1. the multiplicity of independent foci of inflammation, 2. the predominant impairment of the outer layers of the eye and relatively inconsiderable retinal disease, 3. the histamine-like reaction in the epithelium of the conjunctiva and cornea, 4. the tuberculous structure, typical of allergic tissue reaction, 5. the presence of eosinophile cells found frequently in allergic tissue, 6. the negative bacteriological findings, 7. that the disease of the second eye is prevented when the whole source of infection is removed in time, and that excision of the exciting eye is ineffective when the second eye is diseased, 8. sympathetic ophthalmia in cases of evisceration of the exciting eye, 9. meningeal involvement, deafness and bleaching of eyelashes by migration of the virus into other nerve trunks at the base of the brain. Beulah Cushman.

Samuels, Bernard. **The problem of sympathetic Ophthalmia.** Am. J. Ophth. **31**: 397-410, April, 1948. (10 figures.)

Whittington, T. H. **Leucosarcoma of iris.** Pro. Roy. Soc. Med. **65**:65-66, Jan., 1948.

A hemorrhagic yellowish lobed nodule was found protruding from the anterior surface of the iris in a 64-year-old woman. It was not cystic and not transluminable. The tumor was removed by iridectomy and found to be malignant melanoma.

Irwin E. Gaynon.

9

GLAUCOMA AND OCULAR TENSION

Barkan, O. **The technique of goniotomy for congenital glaucoma.** Tr. Am. Acad. Ophth. pp. 210-223, Jan.-Feb., 1948.

The author's streamlining of the older de Vincentii's operation has found its most successful use in some types of congenital glaucoma. Of 76 such operations performed, 66 are reported as having tension normalized, and 10 unsuccessful. The effectiveness of the stripping of the iris from the cornea where it is adherent because of persistent embryonic tissues, depends on several factors. Schlemm's canal must be patent; the cornea must not be so degenerated structurally or functionally that a return to normal is prevented, and the uveal and retinal tissue must not be too badly degenerated to prevent a reasonable return of vision. If the cornea is sufficiently transparent, goniotomy is performed by direct vision under a prismatic contact glass. If not, the operation is performed without a contact glass but with preliminary canthotomy. One-fourth or more of the adherent iris is stripped at one operation which may be repeated, usually without increasing risks. Air or physiological salt solution may be used to further deepen the anterior chamber. This operation, if indicated, should be performed before degenerative changes in the eye doom it to partial or complete failure. Meticulous attention to the numerous details that make goniotomy successful in infantile glaucoma is imperative. Any attempt to abstract these details would be unfair to the author and to the patients. These should be read in the original article by those who are sufficiently deft and understanding to successfully perform this operation.

Chas. A. Bahn.

Cristini, Giuseppe. **Operative results, mechanism of action, and indications for cyclodiathermy in glaucoma.** Giorn. ital. di oftal. **1**:1-13, Jan.-Feb., 1948.

The records of thirty-seven patients operated on by the method of Weekers are reviewed with reference to vision, ocular tension and visual fields (primary glaucoma 18, aphakic glaucoma 3, secondary glaucoma 10, hydrophthalmos 6). In five cases, histopathologic examinations were made of the globes enucleated at varying intervals following the cyclodiathermy.

The change in pressure is attributed by the author to changes in the vascular hydrostatic pressure and of the osmotic pressure and not to variations in the balance between absorption and secretion of the aqueous. The author feels that the Weekers method should not be used in eyes which can be treated by the classic decompression procedures.

Francis P. Guida.

Friede, R. **Treatment of chronic hypotonia bulbi. Wien. klin. Wchnschr. 59**:752-754, Nov. 14, 1947.

The author is interested in attempting to raise the intraocular pressure temporarily (days or weeks) in cases of hypotonia bulbi so that what little vision remains may be preserved and possibly increased and feels that he has succeeded.

He suggests two ways of increasing the intravitreal tension. First, he has the patient drink two or three liters of water for days; and second, he tenotomizes the four recti muscles because he believes the tension at their insertion over the ciliary body tends to decrease the secretion of intraocular fluids by the ciliary body. When the bulb has more tonus he reattaches the muscles. He believes that in early cases of uveitis with hyptotony, the latter may be arrested by his treatment.

Theodore M. Shapira.

Heinz, K. **Retinal detachment following a Lindner bulbus fistula and Elliot trephining.** Proc. Soc. Vienna p. 41, Feb. 22, 1943.

In a man whose left eye had been blind from glaucoma for a long time instillation of homatropine solution into his apparently normal right eye was followed by a severe attack of glaucoma. A Lindner fistula was made and the large quantities of vitreous that appeared were cut off. After 10 days the eye tension was again normal and an Elliot trephining was performed. Four days later the lower half of the retina was found detached but the eye recovered completely after it had been operated upon for this condition.

F. Nelson.

Longhena, Luisa. **Considerations of glaucoma secondary to cataract surgery.** Riv. di oftal. **2**:125-144, March-April, 1947. 1947.

After reviewing the literature the author reports 12 cases of glaucoma following cataract operation. She feels that the glaucoma which follows cataract extraction is not a secondary glaucoma but a primary type, since sooner or later glaucoma appears in the unoperated eye. Repeated tonometric measurements preceding surgery can bring to light this latent glaucoma and help to avoid acute attacks by proper treatment during surgery and post-operatively. Once manifest, glaucoma following cataract surgery must be treated as in a non-aphakic eye. However, the prognosis is poor and frequently in spite of surgical and medical treatment, there is total loss of vision and finally of the eye.

Francis P. Guida.

Matteucci, P. **The transmission of humoral nervous impulses in primary chronic glaucoma.** Ann. d'ocul. **180**:671-680, Nov., 1947.

Quantitatively and qualitatively the aqueous comes from the blood, which in the glaucomatous differs chemically from that of normal eyes. The existence of a central vasomotor disequilibruim in the glaucomatous has been experimentally proven and is becoming more widely accepted. Involved in this disequilibrium is the sympathetic-parasympathetic mechanism. This affects capillary permeability (vasomotor phenomena), also photomotor reactions, and in the glaucomatous may cause a descending optic atrophy. The author's experiments in determining the cholinase and the amino-oxidase in the aqueous and in the blood of normal and glaucomatous patients support the concept that a deficient parasympathetic and/or over-active sympathetic mechanism and reaction exist in the uveal tract of the glaucomatous. For a statistical study of 12 glaucomatous patients and 5 non-glaucomatous patients, the aqueous was obtained at the time of operation for glaucoma or cataract extraction. The data show that the amino-oxidase of normal compared with glaucomatous eyes is higher in the glaucomatous than in the normal eye and also that the cholesterinase in both the serum and aqueous humor is definitely higher in the glaucomatous. Biologic investigators of glaucoma should read this article in the original.

Chas. A. Bahn.

PAN-AMERICAN NOTES

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Communications should reach the editor by the 12th of the month

Pan-American Officers

At the III Pan-American Congress of Ophthalmology, the following officers were elected to serve from 1948 to 1952: President, Dr. Conrad Berens; vice-presidents: Argentina, Dr. Esteban Adrogué; Bolivia, Dr. Aniceto Solares; Brazil, Prof. Ivo Correa Meyer; Canada, Dr. John MacMillan; Chile, Dr. Santiago Barrenechea; Colombia, Dr. Francisco Vernaza; Costa Rica, Dr. Alexis Aguero; Cuba, Dr. Miguel Branly; Ecuador, Dr. Varas Samaniego; Guatemala, Dr. Miguel Medrano; United States, Dr. Frederick C. Cordes and Dr. William L. Benedict. Secretary for north of Panama, Dr. Thomas D. Allen; secretary for south of Panama, Dr. Moacyr E. Alvaro; assistant secretaries for Latin America, Dr. Jorge Balza and Dr. Manoel Silva; assistant secretary for the United States, Dr. Brittain F. Payne; assistant secretary for Central America, Dr. Palomino Dena.

Committees appointed

It was decided at the council meeting and ratified by the general assembly that the following permanent committees should be appointed:

Interamerican Federation of Ophthalmologic Societies. President, Dr. William Benedict (United States); Members: Dr. Jorge Malbran (Argentina), Dr. Aniceto Solares (Bolivia), Dr. Sylvio Abreu Fialho (Brazil), Dr. Italo Martini (Chile), Dr. Carlos Mena (Costa Rica), and Dr. Jorge Suarez Hoyos (Colombia).

Committee on Lighting and Optics. President, Dr. Magin Diez (Argentina); Members: Dr. J. Pascal (United States), Dr. Roque Bellido Tagle (Perú), Dr. J. M. Penichet (Cuba), Dr. Rivas Cherif (Mexico), Dr. A. Cowan (United States), Dr. Caretti (Argentina), and Mr. Tolman (New York).

Committee on Glaucoma. President, Dr. Antonio Torres Estrada (Mexico); Members: Dr. Peter Kronfeld (United States), Dr. Julio Raffo (Perú), Dr. Hilton Rocha (Brazil), Dr. Baudilio Courtis (Argentina), Dr. Esteban Adrogué (Argentina).

Committee for the Encouragement of Research in Ophthalmology. President, Dr. Edwin Dunphy (United States); Members: Dr. Phillips Thygeson (United States), Dr. Esteban Adrogué (Argentina), Dr. Paulo Filho (Brazil), Dr. Lech Junior (Brazil).

Committee on Pharmaceutics. President, Dr. Parker Heath (United States); Members: Dr. Roberto Pereira (Argentina), Dr. K. Swan (United States), Dr. W. Hughes (United States).

Society of "Amigos." Honorary President, Dr. Francisco Belgeri (Argentina). President, Dr. Paulo Cesar Pimentel (Brazil); Members: Dr. William Crisp (United States), Dr. Shaler Richardson (United States), Dr. Magin Diez (Argentina), Dr. Santiago Barrenechea (Chile), Dr. Abelardo Zertuche (Mexico).

Committee on Establishment and Guidance of Ophthalmologic Societies. President: Dr. Jesus Rhode (Venezuela); Members: Dr. Frederick Cordes (United States), Dr. Durval Prado (Brazil), Dr. Roberto Pereira (Argentina), Dr. Oscar Horstmann (Cuba).

Committee on Legal and Industrial Ophthalmology. President, Dr. Jorge Diaz Guerrero (Colombia); Members: Dr. Ralph Lloyd (United States), Dr. Alberto Urrets Zavalia (Argentina), Dr. Santiago Barrenechea (Chile), Dr. J. M. Espino (Venezuela), Dr. Morris Davidson (United States), Dr. José A. Sená (Argentina), Dr. Thomas R. Yanes (Cuba), Dr. Eduardo Arce (Bolivia), Dr. Colombo Spinola (Brazil).

Committee on Standardization of Ophthalmic Hospitals and Clinics. President, Dr. J. H. Dunnington (United States); Members: Dr. Magin Diez (Argentina), Dr. Ivo Correa Meyer (Brazil), Dr. J. M. Penichet (Cuba).

Committee on Interamerican Medical Relationships. President, Dr. R. Pacheco Luna (Guatemala); Members: Dr. Daniel Kirby (United States), Dr. Derrick Vail (United States), Dr. Palomino Dena (Mexico), Dr. Sylvio Abreu Fialho (Brazil), Dr. Georgiana Theobald (United States), Dr. Olga Ferrer (Cuba).

Committee of Purchasing Section. President, Dr. Rene Contardo (Chile); Members: Dr. Paiva Gonçalves (Brazil), Dr. Luis E. de Mora (Perú), Dr. Jorge Malbran (Argentina), Dr. Roberto Vazquez Barriere (Uruguay).

Contact Lens Center Committee. President, Dr. Baudilio Courtis (Argentina); Members: Dr. Arno Town (United States), Dr. Enrique Bertotto (Argentina), Dr. Daniel Silva (Mexico), Dr. J. Pascal (United States).

Committee on Orthoptics. President, Dr. Avery Prangen (United States); Members: Dr. Jorge Malbran (Argentina), Dr. Cesar Rodriguez (Perú), Dr. Raimundo Tartari (Argentina), Dr. J. Mendonça de Barros (Brazil).

Committee on Scientific Cinematography. President, Dr. Hilton Rocha (Brazil); Members: Dr. Gilberto Cepero (Cuba), Dr. A. Bedell (United States), Dr. Magin Puig Solanes (Mexico), Dr. Caretti (Argentina).

Committee on Trachoma. President, Prof. Cesario de Andrade (Brazil); Members: Dr. José R. Toja (Argentina), Dr. Roque Bellido Tagle (Perú), Dr. Paula Santos (Brazil), Dr. Phillips Thygeson (United States).

Bureau of Professors. President, Dr. Jorge Valdeavellano (Perú); Members: Dr. Derrick

Vail (United States), Dr. R. Rodriguez Barrios (Uruguay), Dr. Miguel A. Branly (Cuba), Dr. Alberto Urrets Zavalia (Argentina).

Committee for the Prevention of Blindness. President, Dr. Alberto Vazquez Barriere (Uruguay); Members: Dr. Tomás R. Yanes (Cuba), Dr. Sanchez Bulnes (Mexico), Dr. Natalicio de Farias (Brazil), Dr. Magin Diez (Argentina), Dr. C. Espildora Luque (Chile), Dr. Franklin Foote (United States).

Committee on Statutes. Dr. Tomás R. Yanes, Dr. Moacyr E. Alvaro, Dr. Conrad Berens, Dr. Alberto Vazquez Barriere, Dr. Thomas D. Allen, Dr. Santiago Barrenechea.

Board of Censors. President, Dr. Tomás R. Yanes (Cuba); Members: Dr. Alberto Vazquez Barriere (Uruguay), Dr. Esteban Adrogué (Argentina), Dr. Enrique Cipriani (Perú), Dr. Derrick Vail (United States), Dr. Rene Contardo (Chile), Dr. Paulo C. Pimentel (Brazil), Dr. Luis Sanchez Bulnes (Mexico), Dr. Constantino Herdocia (Costa Rica).

Committee on Neuro-Ophthalmology. President, Dr. Alejandro Posada (Colombia); Members: Dr. Pedro Falcão (Brazil), Dr. Jorge Malbran (Argentina), Dr. Esteban Adrogué (Argentina), Dr. Frank Walsh (United States), Dr. Alfred Kestenbaum (United States).

NEWS ITEMS

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News items should reach the editor by the 12th of the month

DEATHS

Dr. Archibald Alexander MacLachlan, Pittsburgh, Pennsylvania, died December 2, 1947, aged 73 years.

Dr. Hamilton Stillson, Seattle, Washington, died January 11, 1948, aged 90 years.

Dr. George A. Morley, Crookston, Minnesota, died recently, aged 79 years.

MISCELLANEOUS

REPORT OF EYE-BANK

The Stanford Eye Bank has reported that it provided 33 corneas for eye-graft operations during its first year of operation. All qualified eye surgeons on the Pacific coast are invited to make use of the facilities of the bank. Corneas collected and preserved by the Eye-Bank are distributed without cost to either patient or surgeon, except for a small service fee. Information on the correct procedure for registration of donors can be obtained by writing to the Stanford Eye Bank, 2398 Sacramento Street, San Francisco.

RECOGNITION DINNER HELD

On April 22, 1948, a recognition dinner was held at Talladega, Alabama, following the inauguration earlier this year of the assumption by the Department of Ophthalmology, Medical College of Alabama, of the full ophthalmic responsibility for the Alabama Institute for the Deaf and Blind. Representative officials of the state and of the university were present. There are 209 blind or partially blind children at the institute and much investigation and therapeusis are planned, particularly with reference to those children with progressive ocular disease. A resident in ophthalmology spends two days each week at the institute (50 miles from Birmingham) and the rest of his time at the Medical College and the Thigpen-Cater Eye Hospital. Dr. Arthur Steinmetz, Hayward, California, is the present resident.

SOCIETIES

The Milwaukee Oto-Ophthalmic Society held its regular meeting on April 27th at the Milwaukee Children's Hospital. The scientific program included: "Surgery of strabismus with special reference to the oblique muscles," Dr. Beulah Cushman, Northwestern University, Chicago; and "Treatment of acute laryngo-tracheo-bronchitis," Dr. Howard High, Milwaukee.

PENNSYLVANIA ACADEMY MEETS

The Pennsylvania Academy of Ophthalmology and Otolaryngology met on April 23rd, 24th, and 25th at the Penn-Harris Hotel, Harrisburg. Dr. Gabriel Tucker was the guest of honor.

Of particular interest to eye physicians were the following papers: "The surgery of the extraocular muscles," Dr. Glen Gregory Gibson, Philadelphia; "Retinal detachment," Dr. John H. Dunnington, New York; "Surgery of the lids and conjunctiva," and " External ocular diseases and their treatment," Dr. Raymond Emory Meek, New York; "Integrated implants and the presentation of a new type universal implant," Dr. Norman L. Cutler, Wilmington, Delaware; "General anesthesia for operations on the eye, ear, nose, and throat," Dr. Philip D. Woodbridge, Reading, Pennsylvania; and "Pitfalls in cataract surgery," Dr. Carroll R. Mullen, Philadelphia.

Two splendid motion pictures of ophthalmic interest were shown. They were "Keratoplasty," and "Cataract extraction—Extracapsular, intracapsular with forceps, intracapsular by suction." Both were made by Dr. Ramon Castroviejo, New York.